AD-A104 351 AIR FORCE SYSTEMS COMMAND WASHINGTON DC F/G 17/9
DRAFT SUPPLEMENT TO FINAL ENVIRONMENTAL STATEMENT ON CONTINENTA--ETC(U)
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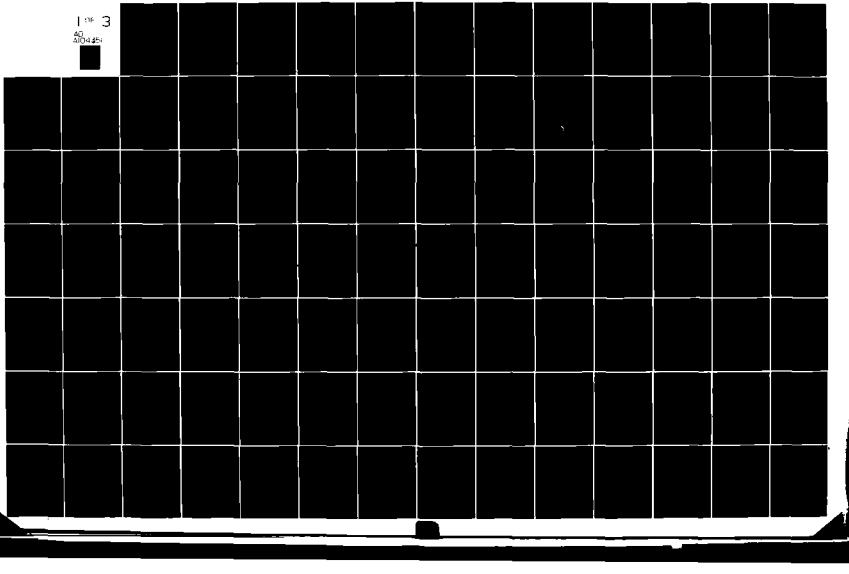

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SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

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1. REPORT NUMBER: AFSC-TR-81-62	2. GOVT ACCESSION NO.: AD-A104 351
3. RECIPIENT'S CATALOG NUMBER: 13	
4. TITLE (and Subtitle): Draft Supplement to Final Environmental Statement on Continental United States (CONUS) Over-the-Horizon Backscatter (OTH-B) Radar System, Penobscot, Washington, Aroostook County, Maine	5. TYPE OF REPORT & PERIOD COVERED: Draft 1981
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): Stanford Research Institute International Palo Alto CA	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS: Electronic Systems Division (ESD/OCU) Hanscom AFB MA 01731	12. REPORT DATE: August 1981
	13. NUMBER OF PAGES: 226
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)	15. SECURITY CLASS. (of this report): Unclassified
	15a. DECLASSIFICATION DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Unclassified Unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

CONUS OTH-B
OTH
Supplemental DEIS

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

See reverse

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Department of the Air Force
Air Force Systems Command
Electronic Systems Division

CONTINENTAL UNITED STATES (CONUS)
OVER-THE-HORIZON BACKSCATTER (OTH-B) RADAR SYSTEM
PENOBSCOT, WASHINGTON, SOMERSET COUNTIES, MAINE

DRAFT SUPPLEMENT--ENVIRONMENTAL IMPACT STATEMENT

ABSTRACT
This supplement amends the Final Environmental Impact Statement issued in January 1975. The action proposed in the 1975 EIS was to construct and operate an OTH-B radar system in Maine, initially covering a 60 degree sector, and later expanded to cover 180 degrees. The action proposed in this supplement is to locate the integrated operations, maintenance, and security facility at Bangor International Airport, a location not considered in the 1975 EIS. Alternatives are locating at Bucks Harbor the maintenance and security personnel who support the Washington County receiver site, but leaving the maintenance and security personnel for the operations center and the Somerset County transmitter site with the operations personnel at Bangor International Airport; and not deploying either the 60 degree or the 180 degree system. The scope of the supplement is limited mainly to identifying the environmental consequences of the operations center alternatives and examining the issue of biological effects of nonionizing radiation from the remote Somerset County transmitter on the basis of data available after the 1975 EIS was completed. Both biophysical and socioeconomic consequences of the operations center alternatives were found to be small. Further examination of the biological effects issue confirmed the conclusions of the 1975 EIS. No reliable evidence has been found to indicate that any hazard will result from either short-term or prolonged exposure of people to the power densities of the Somerset County OTH-B transmitters outside the site exclusion fences.

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SUMMARY

DRAFT SUPPLEMENT--ENVIRONMENTAL IMPACT STATEMENT

Over-the-Horizon Backscatter (OTH-B) Radar System
Penobscot, Washington, Somerset Counties, Maine

This supplement amends the Final Environmental Impact Statement (EIS) issued January 1975. The action proposed in the 1975 EIS was to construct and operate an OTH-B radar system in Maine, initially covering a 60 degree sector, and later expanded to cover 180 degrees. The action proposed in this supplement is to locate the integrated operations, maintenance, and security facility at Bangor International Airport, a location not considered in the 1975 EIS. Alternatives are to locate at Bucks Harbor the maintenance and security personnel who support the Washington County receiver site, while leaving the maintenance and security personnel for the operations center and the Somerset County transmitter site with the operations personnel at Bangor; and not to deploy either the 60 degree or 180 degree system. The scope of the supplement is limited mainly to identifying the environmental consequences of the operations center alternatives, and examining the issue of biological effects of nonionizing radiation from the remote Somerset County transmitter on the basis of data available after the 1975 EIS was completed.

Other than the alternative not to deploy the system, which obviates the socioeconomic and biophysical consequences of the other alternatives, no alternative stands out clearly as environmentally preferred. Because the proposed action to base all personnel at Bangor International Airport involves a 10% smaller total staff than the Bucks Harbor alternative, and affects one site instead of two sites, it may be judged to be slightly preferred on environmental grounds.

Bangor

Socioeconomic Consequences. The new employees and dependents associated with the proposed action are expected to live in existing houses in Bangor or in other communities within the region of influence (ROI). The present school system and other community services are adequate and will be able to accommodate the new population. No induced growth or development is anticipated. Benefits would accrue to the ROI in the way of increased income and spending.

Biophysical Consequences. The construction of the operations center and support facilities on land currently used by the Air National Guard and undeveloped city-owned land at Bangor International Airport would not adversely affect the local terrestrial and aquatic ecosystems (e.g., plants, animals, and soils). Nor would regional ecological

conditions be affected by the new population associated with the operations center because no housing or additional offsite construction is expected in the region.

Air pollutants would be emitted at the operations site by an auxiliary generator and by employee automobiles. The additional population would cause small amounts of pollutants to be emitted throughout the ROI from automobile travel. These emissions would have a minor effect on existing air quality. The airport is a clean air area, and the entire new population is unlikely to reside in central Bangor, where levels of some pollutants have exceeded standards.

Local surface water quality would be affected only in the event of accidental leakage from the fuel storage tanks at the operations site. The Bangor water supply and sewage disposal systems are adequate to accommodate all new demand associated with the proposed action. Noise levels at the airport and in the region as a whole are not expected to be altered noticeably by the activities related to the proposed action.

Bucks Harbor Alternative

Socioeconomic Consequences. To accommodate the personnel associated with the alternative facility at Bucks Harbor, new housing units would be constructed on the station. Noticeable impacts on existing community services, including schools, are not likely. Communities in the region would benefit from an increase in expenditures and additional income.

Biophysical Consequences. No significant biophysical impacts are anticipated in the Bucks Harbor area as a result of the alternative of constructing maintenance and security facilities and locating associated personnel and their dependents there. The nesting bald eagles in Machias Bay are not likely to be disturbed by the additional activity. Air emissions, sewage (new septic systems may have to be constructed), and noise would increase slightly.

Transmitter Site, Somerset County

The conclusion of the 1975 EIS regarding health effects is unchanged: no reliable evidence has been found to indicate that any hazard would result from either short-term or prolonged exposure of people to the power densities from the Somerset County OTH-B radar transmitters outside the site exclusion fences. This supplement provides extensive information on the topic. The conclusion is based on 1) an assessment of the scientific literature, 2) a comparison with current and proposed standards, and 3) the remote location of the transmitter site.

In addition, the safe separation distances for persons handling electroexplosive devices (EEDs) are revised downward in recognition of

the lower power densities of the system now planned (25% of the power density originally planned). Because of both lower transmitted power and improved pacemaker shielding, no significant risk to cardiac pacemaker wearers is now expected outside the exclusion fences that prevent personnel from entering the areas close to the antennas when the radar is operating.

No Action Alternative

If the system is not deployed, the socioeconomic and biophysical consequences discussed above would not occur. The experimental radar system presently deployed would be dismantled and removed, and the OTH-B program would be terminated. The benefits to the security of the nation from operating the OTH-B radar would not accrue.

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Section 1

INTRODUCTION

This supplement amends the 1975 final Environmental Impact Statement (1975 EIS) for the Continental United States (CONUS) Over-the-Horizon Backscatter (OTH-B) Radar System (Air Force, 1975). This supplement focuses on the effects of certain changes in plans for the system and does not repeat the analysis or conclusions for aspects of the OTH-B system that are substantially unchanged.

This section briefly describes the purpose and need for the radar system, activities related to the system to date, and planned modifications in the system that necessitate this supplement. It also defines the scope of the supplement. Section 2 describes the affected environment, and Section 3 describes the environmental consequences related to the planned changes in the system. Section 4 describes the affected environment and environmental consequences for the alternatives to the proposed action. Subject areas not affected by planned system changes and, hence, not reassessed in this supplement include: adverse effects which cannot be avoided, relation between short-term use and long-term productivity, and irreversible or irretrievable commitments of resources.

1.1 System Purpose and Need

1.1.1 System Mission

Currently, airspace surveillance and tactical warning of a potential bomber attack against the North American continent is provided by a series of line-of-sight radar systems with limited detection range. Although this radar surveillance system has helped deter a potential bomber attack to date, its range and altitude limitations are not adequate to provide the necessary tactical warning against the improved capabilities in aircraft and weapons delivery systems of potential adversaries. Increased tactical warning time beyond that provided by line-of-sight radars is urgently needed.

The OTH-B radar system, with the ability to detect aircraft at any altitude approaching the North American continent at distances of approximately 1,800 nautical miles, can provide this increased tactical warning. Figure 1-1 shows the planned deployment of this system. Such early detection of a bomber attack can provide the National Command authorities increased decision time to enhance the survivability of retaliatory forces and to redeploy available defense forces.

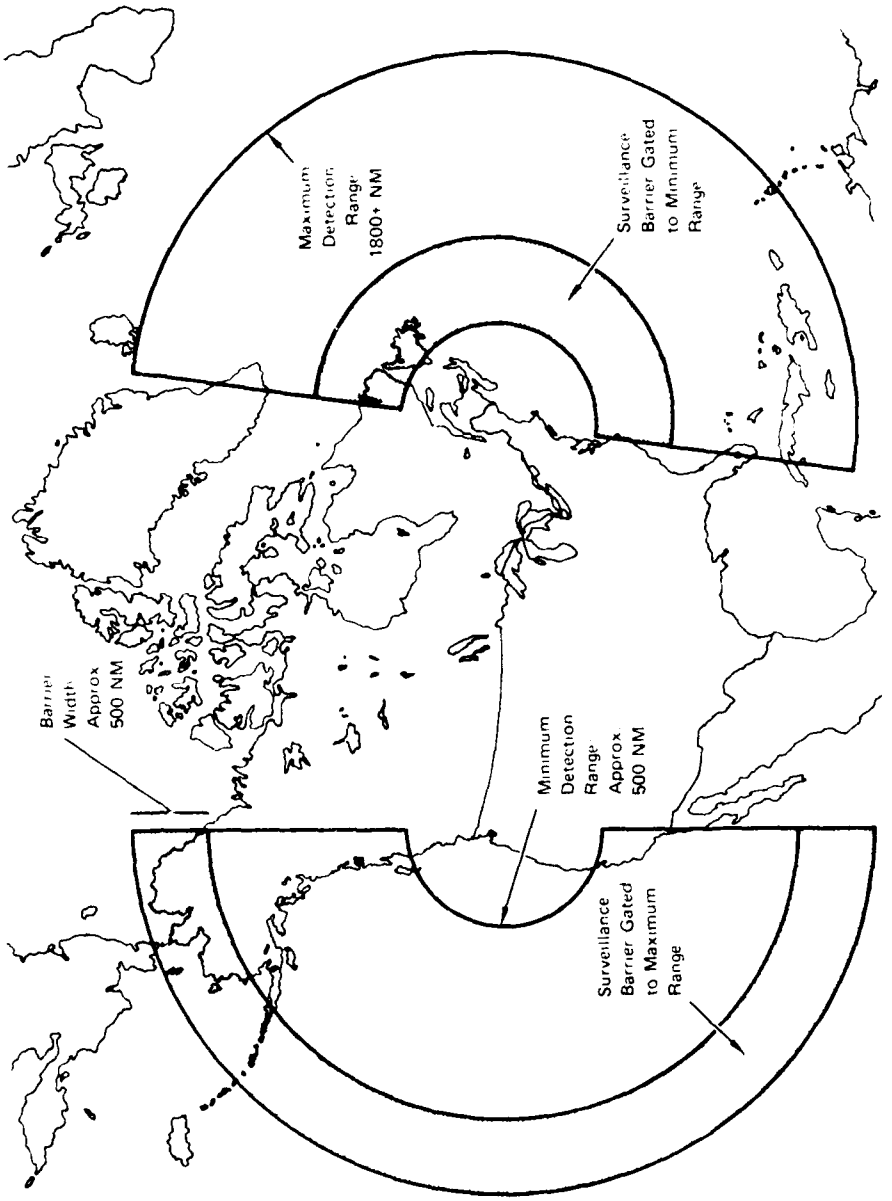

FIGURE 1-1 REPRESENTATIVE CONUS OTH-B RADAR BARRIER COVERAGE

1.1.2 Background

Two OTH-B operational radar systems (ORS) are planned, one near each coast (see Figure 1-1). Each ORS will cover 180 degrees in azimuth and will include 3 transmitter and 3 receiver sectors, each sector covering 60 degrees in azimuth. The transmitter and receiver antennas and equipment will be located at remote sites, separated by about 100 nautical miles (nm) to prevent interference. Processed data will be communicated from the receiver location to an operations site for correlation with known aircraft positions. Subequently selected data will be forwarded to designated locations for further action.

Barrier coverage nominally 500 nm in range extent will be provided. The barrier will be movable in range to provide coverage from a minimum of approximately 500 nm from the radar transmitter locations to a maximum of approximately 1,800 nm from each location (see Figure 1-1). The northernmost edge of the barrier coverage will be located to satisfy operational needs.

The radar signals are transmitted around the earth's curvature by refraction of the signal in the electrically charged ionosphere, an atmospheric stratum above the stratosphere. Energy reflected from targets is returned to the receiver location by the same process. Propagation characteristics using ionospheric refraction permit detection of targets at all altitudes beyond the horizon and, therefore, well beyond the detection capability of ground based, line-of-sight radars.

The operations site is the locus for operation and control of the associated transmitters and receivers. External data required for proper radar operation and identification of targets are entered into the system at each operations site, e.g., aircraft position reports, ionospheric conditions data, and other similar information. Appropriately formatted messages are released from each operations location and forwarded to National Command authorities.

The OTH-B radar program was started in the early 1970s. In 1975, a contract was let to build a prototype radar system (PRS), intended to prove out engineering concepts. Specifically, the PRS was to have been built to demonstrate both the technical feasibility and operational suitability of the OTH-B radar.

The PRS was to have provided 60 degrees of azimuthal coverage, effectively comprising one sector of the ORS. If the PRS were successful, the program was to proceed with the ORS. The environmental impact of the proposed system was assessed, meetings with federal, state, and local officials were held, and a final Environmental Impact Statement (EIS) was released in January 1975 (Air Force, 1975). The 1975 EIS covered the construction and operation of the PRS and the ORS for the East Coast. The EIS was prepared in accordance with Council on

Environmental Quality (CEQ) and Air Force guidelines and regulations in force at that time. The impacts of subsequently constructing and operating the West Coast system were to be covered in a separate EIS.

The prototype system described in the 1975 EIS was not actually installed. Budget limitations dictated the construction of a smaller version, termed an Experimental Radar System (ERS), with limited frequency coverage--6.74-22.25 MHz instead of 5-30 MHz--smaller azimuthal coverage--30 degrees instead of 60 degrees--and reduced power--1.2 megawatts (MW) instead of 2.4 MW. The ERS was intended to demonstrate only technical feasibility of the OTH-B radar, the operational suitability of the program could not be assessed. Because the environmental consequences of the ERS would be no more adverse, and in some cases would be less adverse, no further documentation of environmental effects was made when the ERS was installed.

The Air Force now intends to upgrade the ERS to a full-scale, 60-degree sector very similar to the original prototype to establish operational suitability, and then to implement the original plan to expand that 60-degree sector by adding two similar sectors to cover the full 180 degrees in azimuth. A comparison of the plans outlined in the 1975 EIS with current plans revealed the need to prepare and issue this supplement to the 1975 EIS.

1.1.3 Need for Supplement and Scope

The need for this supplement arises from two major considerations:

- A change in the planned siting of a part of the system that affects the location of operating, maintenance, and security personnel
- A change in the public perception of the potential significance of biological effects of radiofrequency (nonionizing) radiation.

No other information is available that would indicate the need for further amendment of the EIS at this time. For example, the environment at the remote transmitter and receiver sites is substantially unchanged, and no substantial changes in land use in the vicinity have been noted. Consequently, the scope of this supplement is limited mainly to evaluation of the socioeconomic and related biophysical impacts associated with the location of the operating, maintenance, and security personnel at currently proposed and alternative sites, and an examination of the issue of biological effects based on data more recent than those used in the 1975 EIS. Both the initial 60-degree sector and the full 180-degree system are addressed in this supplement, as they were in the 1975 EIS.

1.2 Changes to the Proposed Action and Alternatives

The sites of the action proposed in the 1975 EIS are shown in Figure 1-2. The transmitter and receiver sites were to be located in remote areas near Moscow and Columbia Falls, respectively. The sites initially proposed for the operations center were at Loring Air Force Base, Bucks Harbor, and Topsham. The ERS transmitter site and collocated receiver/ operations site were installed near Moscow and Columbia Falls as planned for the prototype. (The receiver and operations sites were collocated for economic reasons.) However, further evaluation and planning have led the Air Force to eliminate the three original sites from consideration as operations center locations for the full 180-degree system for the following reasons: overall cost, proximity to both transmitter and receiver sites for integrated maintenance and security support, and availability of existing military base and community support facilities.

1.2.1 Operations Site Location and Alternatives

The location now proposed for the integrated operations center and maintenance and security facility is the Bangor International Airport (BIA), formerly Dow Air Force Base. The location is shown in Figure 1-2. Specifically, the building housing the integrated operations center and support facilities would be located within the airport boundary on a federally owned parcel of 338 acres that is now used by the Air National Guard and on an 11-acre parcel to be acquired from the city of Bangor. Support facilities include a commissary, base exchange, dining hall, dormitory, gymnasium, recreation center, and like facilities. Bangor is near the center of the state and is approximately midway between the transmitter and receiver sites. The BIA is on the western side of the city.

An alternative to locating support personnel (maintenance and security) for the operations center, transmitter site, and receiver site at BIA is to locate the support personnel for the receiver site at Bucks Harbor, and the support personnel for the operations center and transmitter sites along with operations center personnel at BIA. The Bucks Harbor Air Force Station is situated on a peninsula on the southeastern coast of Maine, west of Machias Bay (see Figure 1-2).

Another alternative is to discontinue plans to enlarge the ERS to implement the East Coast OTH-B system. This alternative is similar to the no-action alternative in the 1975 EIS, except that the ERS which has been built would be dismantled and removed.

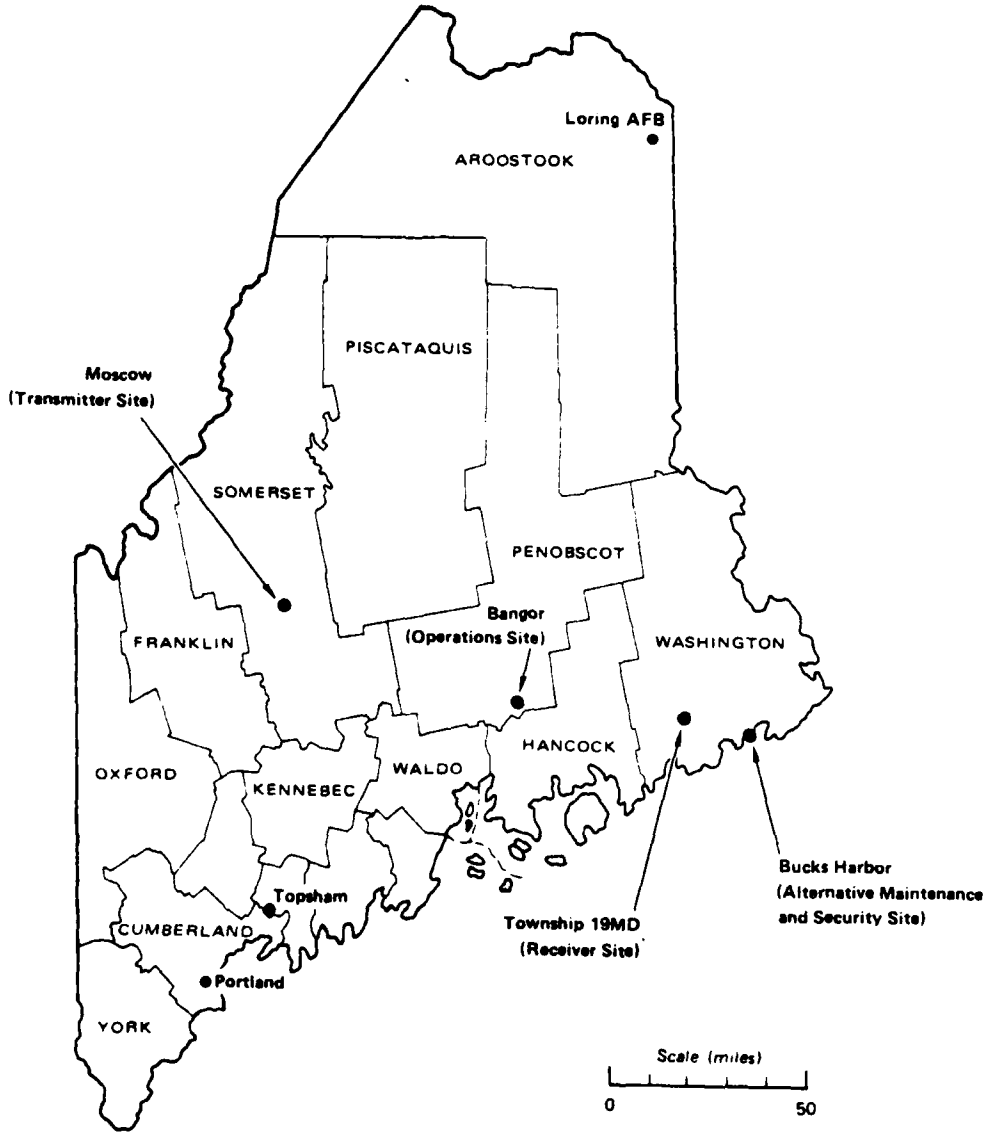

FIGURE 1-2 SITE LOCATIONS

1.2.2 Transmit Site Configuration and Specifications

Areas currently being considered for locating the second and third 60-degree sectors are shown in Figure 1-3, along with the ERS site that will be expanded to construct the first sector. All are in the immediate vicinity of the area planned and shown in the 1975 EIS.

Both the maximum transmitter power and the antenna gain now planned for each sector are 50 percent as large as those specified for the prototype and described in the 1975 EIS. Consequently, the maximum power density will be 25 percent as large as originally planned. Detailed information on the maximum power densities expected on the ground and in the air is given in Appendix A. This description provides the basis for the assessments of potential adverse effects of the transmitter's operation detailed in Appendices B and C, and summarized in Sections 3.2 and 3.3.

1.2.3 Receiver Site

The area currently being considered for locating the second and third 60-degree sectors is shown in Figure 1-4, along with the ERS site that comprises the first sector. The planned location is still in the vicinity of the ERS receiver site, an uninhabited area in townships 18 and 19 Middle District within Washington County, and is consistent with the option 1 alternative in the 1975 EIS. However, a 325-acre parcel identified as productive blueberry land in the 1975 EIS, and part of option 1, is now not required. Consequently, no commercial blueberry land will be needed to expand the system to 180-degree coverage, and the 650 acres of commercial blueberry land estimated to be removed from production in the 1975 EIS is effectively reduced to the 325 acres already removed in constructing the ERS.

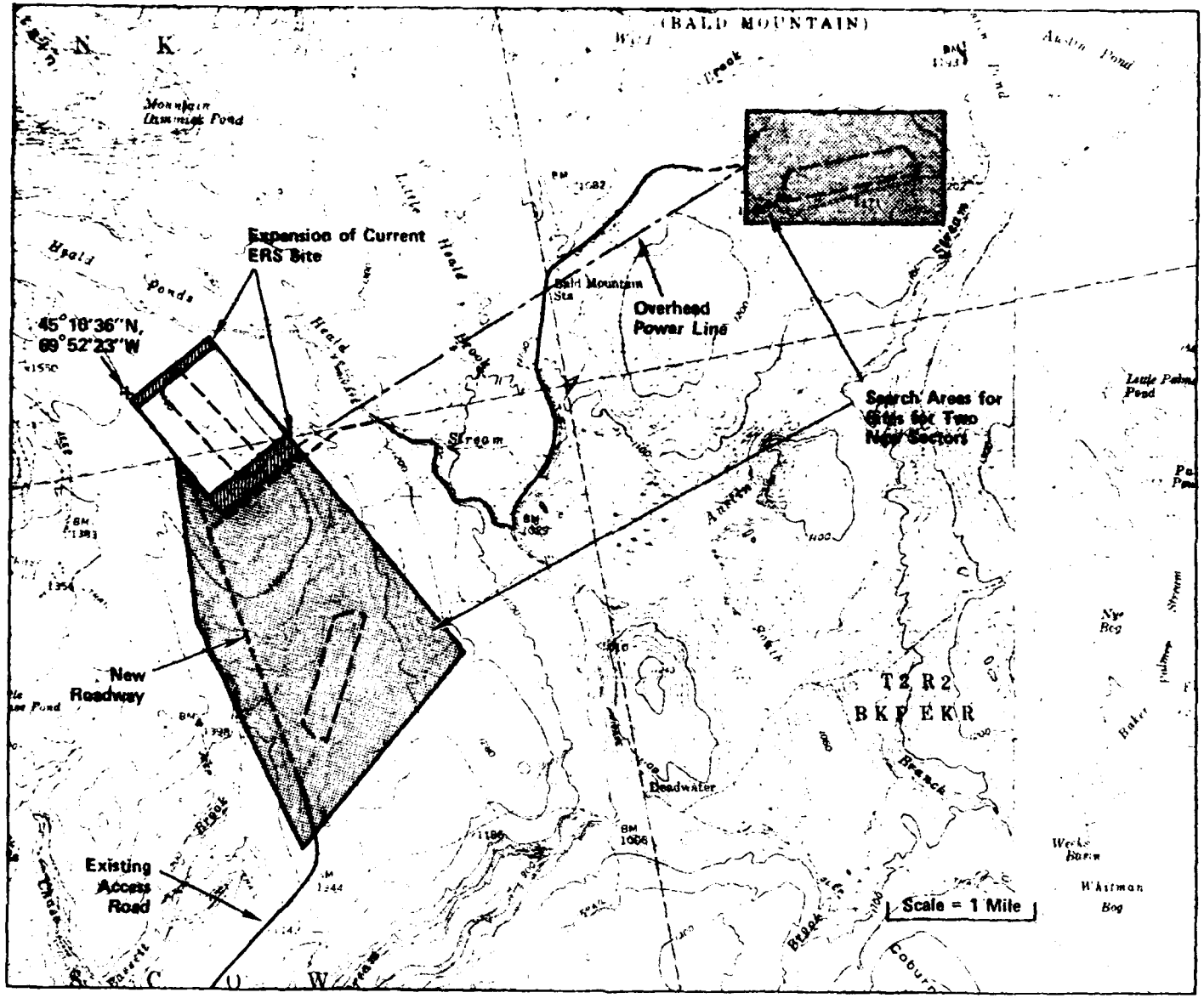

FIGURE 1-3 TRANSMITTER SITE LOCATIONS

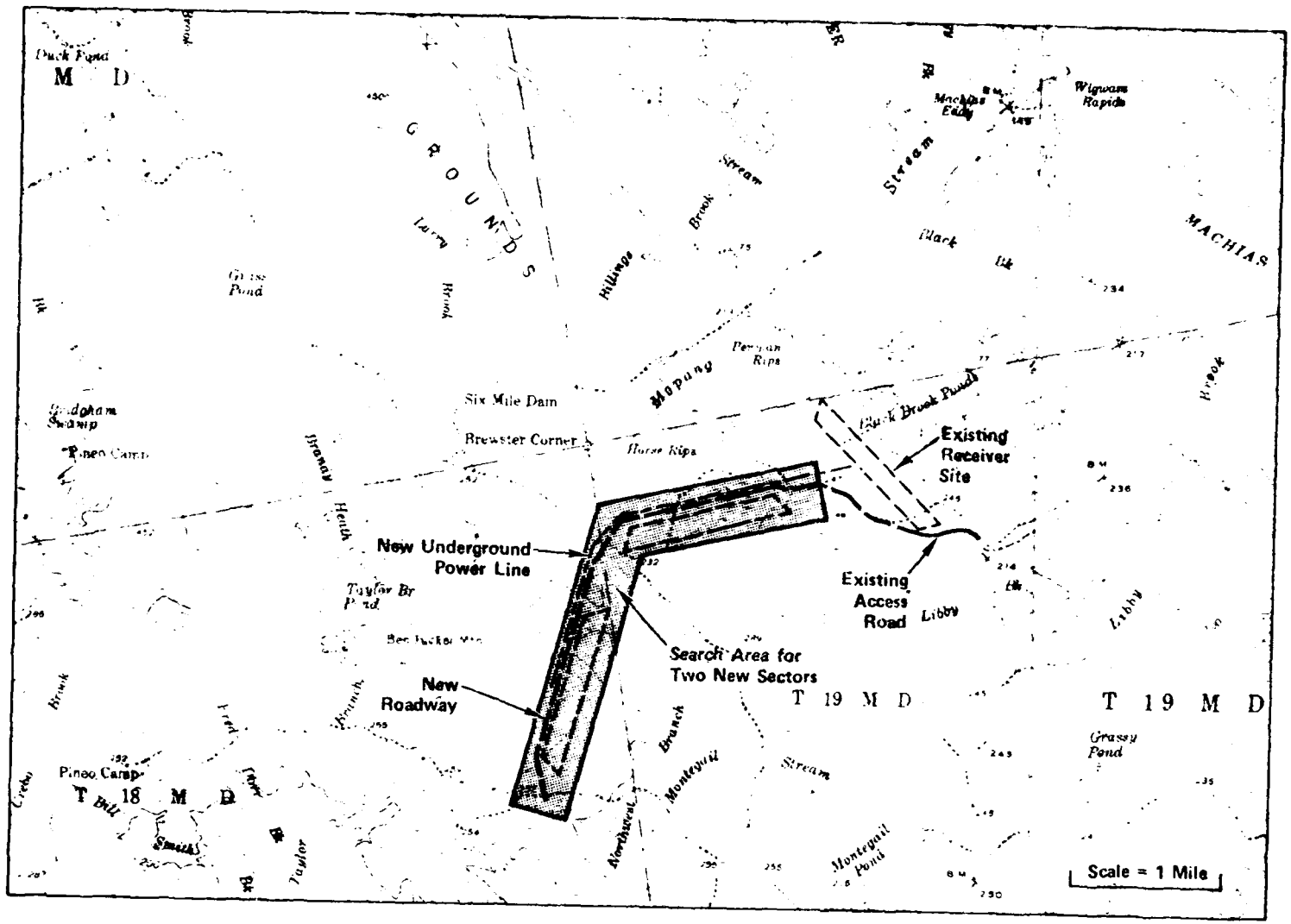

FIGURE 1-4 RECEIVER SITE LOCATIONS

Section 2

UPDATE OF THE ENVIRONMENT AFFECTED BY THE PROPOSED ACTION

This section describes the socioeconomic and biophysical environments at the Bangor site proposed for the integrated operations, maintenance, and security facilities, and it provides an update on the transmitter site in Somerset County. The Bangor site is affected by both the proposed action and the Bucks Harbor alternative. The environment at Bucks Harbor affected by the Bucks Harbor alternative is described in Section 4.

2.1 Affected Socioeconomic Environment--Bangor

Bangor is located on the Penobscot River, 35 miles southeast of the geographical center of Maine and in south-central Penobscot County. Bangor International Airport (BIA), the proposed site of the OTH-B radar system operations center and integrated maintenance and security facility, is located in the western portion of the City of Bangor (see Figure 2-1). Until 1968, the airport was Dow Air Force Base; some land within the airport is still reserved for military use by the Air National Guard and the Army National Guard. The operations center and its support facilities would be constructed on a site within a 338-acre parcel currently under the jurisdiction of the Air National Guard, and on an 11-acre site to be acquired from the city of Bangor.

Bangor is the largest city in Penobscot County and serves as a market and employment center for much of central Maine. This analysis of existing socioeconomic conditions and potential impacts focuses on Bangor and ten surrounding communities, i.e., the Region of Influence (ROI). The ROI, as shown in Figure 2-2, consists of the communities of Bangor, Brewer, Eddington, Glenburn, Hampden, Hermon, Holden, Old Town, Orono, Orrington, and Veazie. These communities are within 10 miles to 15 miles of the proposed operations center and provide the majority of the labor force for Bangor. Collectively, they comprise the Bangor-Brewer Labor Market Area (LMA). The majority of Air Force and civilian personnel assigned to the operations center are likewise expected to reside in the Bangor ROI.

2.1.1 Employment

The Bangor ROI had a labor force of 40,800 in 1980 and unemployment averaged 2,875 persons (Maine Department of Manpower Affairs, 1981a). The unemployment rate averaged 7.0 percent in 1980, up from 6.2 percent in 1979. In comparison, the unemployment rate in 1980 was 7.7 percent for Maine and 7.1 percent for the United States (Maine Department of Manpower Affairs, 1981b; Department of Commerce, 1981).

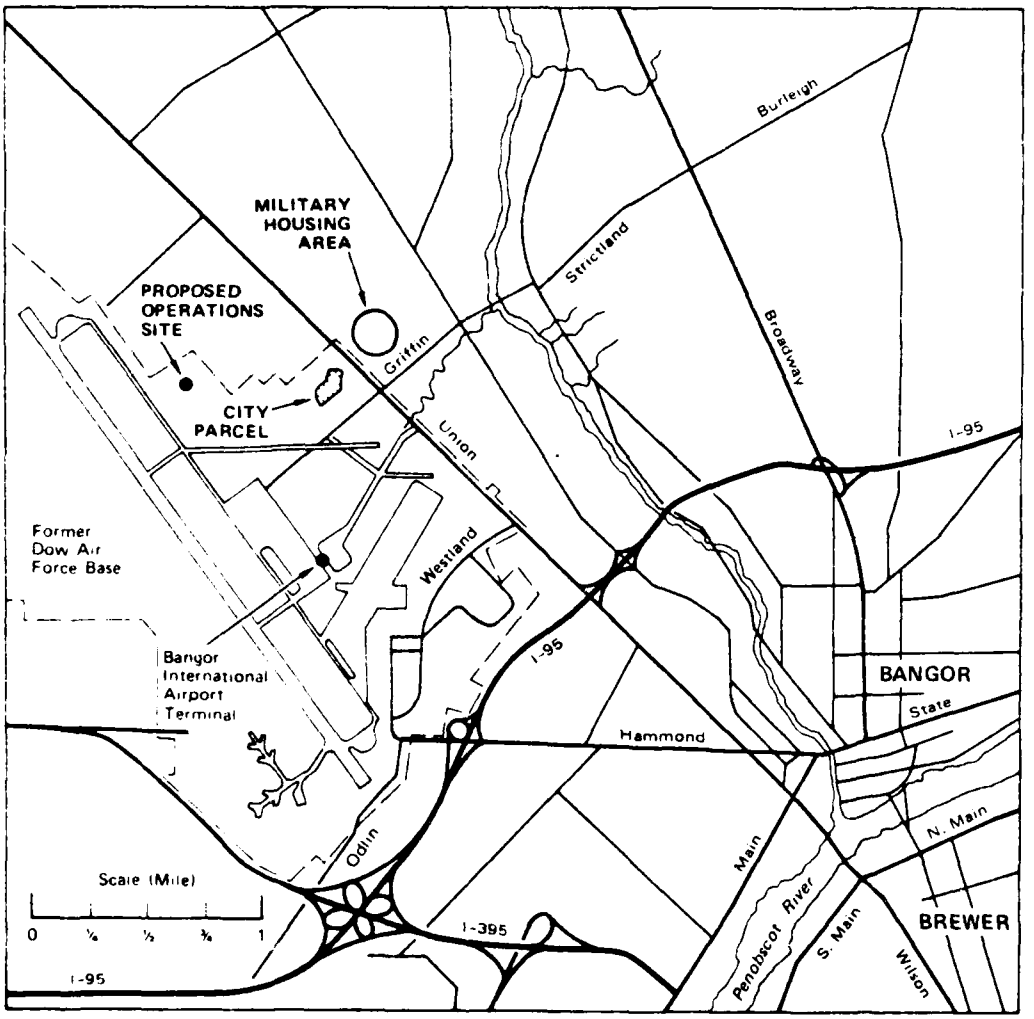

FIGURE 2-1 PROPOSED OPERATIONS SITE AND BANGOR-BREWER AREA

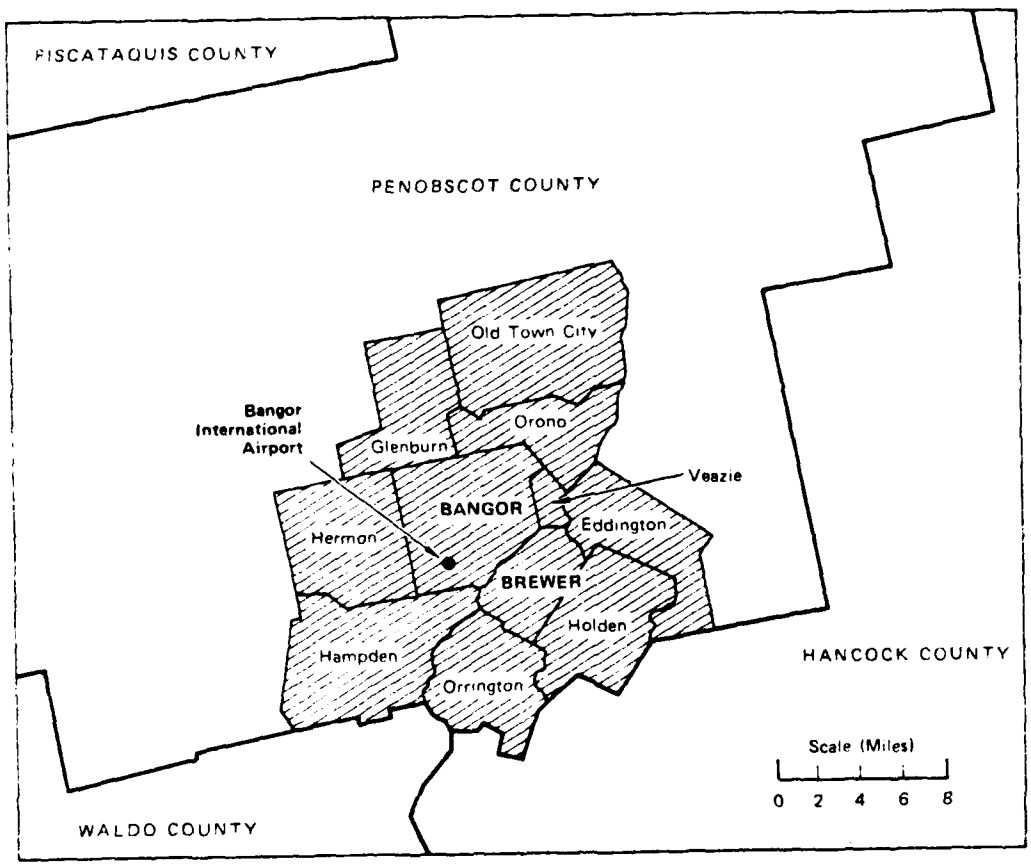

FIGURE 2-2 REGION OF INFLUENCE (BANGOR-BREWER LABOR MARKET AREA) FOR THE PROPOSED OPERATIONS CENTER

The current ratio of labor force to population in the ROI is 0.51. If this ratio remains constant through 1985, the labor force would be 41,300 persons in 1985.

Three sectors account for 75 percent of employment in the Bangor ROI:

Wholesale and retail trade	24 percent
Services	25 percent
Government	26 percent

Manufacturing accounts for 14 percent of employment in the ROI.

2.1.2 Population

The population of the 11 communities in the Bangor ROI totaled approximately 80,000 in 1980, a 3.4 percent increase from the 1970 population. As shown in Table 2-1, growth trends varied considerably among the communitites. The populations of Bangor and Brewer decreased by 4.6 percent and 3.1 percent, respectively, whereas those of Hermon and Holden increased by more than 33 percent. The population of the town of Glenburn increased by approximately 1,100 persons, or more than 90 percent. This reflects the trend toward residential growth on the periphery of Bangor.

If the population of the ROI continues to grow at the rate it did between 1970 and 1980, the population would be approximately 81,000 in 1985 and 82,200 in 1990.

Table 2-1

POPULATION IN THE BANGOR ROI

	Population		Percent Change
	1970	1980	1970-80
Bangor	33,168	31,645	-4.6
Brewer	9,300	9,010	-3.1
Eddington	1,358	1,770	30.3
Glenburn	1,196	2,315	93.5
Hampden	4,693	5,246	11.8
Hermon	2,376	3,162	33.1
Holden	1,841	2,551	38.6
Old Town	8,741	9,026	3.3
Orono	9,989	9,958	-0.3
Orrington	2,702	3,245	20.1
Veazie	1,556	1,612	3.6
Total	76,920	79,540	3.4

Source: Department of Commerce, Bureau of the Census, 1980

2.1.3 Income

Per capita income in 1979 averaged $6,734 in the ROI, compared with $7,052 for Maine. From 1974 through 1979, per capita income grew at an average annual rate of 9.9 percent; however, if the figures are adjusted for inflation, growth in per capita income averaged only 1.7 percent per year. Per capita personal income in 1980 dollars is estimated to be $7,800 in 1980; if historical growth rates continue, it is projected to be $8,500 (1980 dollars) in 1985.

In 1979, total personal income in the Bangor ROI is estimated to be $534 million. Total personal income in the Bangor ROI is projected to be approximately $690 million (1980 dollars) in 1985.

2.1.4 Housing

According to the 1980 census, there are 29,400 housing units in the Bangor ROI (Department of Commerce, Bureau of the Census, 1980). The number of units grew by 21 percent between 1970 and 1980. This growth is a result of the combined effects of a 3.4 percent increase in population and a decrease in the average number of persons per unit, from 3.16 in 1970 to 2.71 in 1980. For Maine as a whole, the average number of persons per housing unit was 2.24 in 1980. The decrease in persons per unit is expected to continue, although at a slower pace, because the rate of household formation is expected to decline, and financing for housing is expected to continue to be expensive.

Assuming the population of the ROI increases to 81,000 persons in 1985 and the number of persons per unit remains at 2.71, the total number of units would increase by only 600 units, for a 1985 total for the ROI of 30,000. If one extrapolates from historical trends, the 1985 projection would be more than 32,000 units. Actual growth will probably fall between these extremes--30,000 units will be used as a lower bound to estimate impacts in 1985.

In 1979, the vacancy rates for the city of Bangor were 4.4 percent for owner-occupied units and 3.7 percent for rental units. In a stock of 9,810 units suitable for occupancy, 238 owner-occupied units and 164 rental units were available, or 402 units altogether. About 200 units suitable for rehabilitation were also vacant. At the BIA 165 units of military housing will be available, and construction of dormitory facilities including 140 beds is planned.

Vacancy rates for the ROI are not available. If it is assumed that the vacancy rate in Bangor is indicative of that in the ROI, another 800 units are vacant, for a total of approximately 1,200 vacant units in the Bangor ROI.

2.1.5 Educational Facilities

The number of pupils residing in the Bangor ROI was approximately 14,700 in the 1980-1981 school year (Fish, 1981). The ratio of students

to total population is 0.17. School enrollments in the ROI have decreased in recent years.

The Bangor public school facilities include 1 high schoool, 3 middle schools, and 8 elementary schools. The Bangor School Committee voted to close the Union Street Middle School after this school year due to a continuing decline in enrollment in the system. Similarly the committee was previously forced to close the 14th Street elementary school. Although it is presently closed, the 14th Street school is being held in reserve by the school committee in case student enrollments should increase in the future.

The total projected enrollment in the Bangor school system for the academic year 1981-1982 is projected to be 4,650 students. The average pupil to teacher ratio in the Bangor school system for Kindergarten through grade 12 is projected to be 19.5 to 1. During the 1980-1981 school year the kindergarten through eighth grade pupil/teacher ratio was 23 to 1.

2.1.6 Community Facilities and Services

Publicly provided facilities and services in Bangor are adequate for the population of the region. Police, fire, and medical services are readily available.

2.1.7 Land Use

The land surrounding Bangor is characterized by rolling hills and forests, as well as lakes and streams. The land that is suitable for agriculture contains highly productive dairy, poultry, and potato farms. Recreational use of the land is also common in the local area. Industrial uses of the land have included pulp and paper mills, shoe manufacturing facilities, and, more recently, electronics plants.

Bangor has 12 industrial parks, 2 of which are located at BIA. Their proximity to the operations site is shown in Figure 2-3. One of these industrial parks has approximately 85 acres available for development, in parcels ranging in size from 2 to 15 acres. The other park includes 125 acres available for industrial manufacturers, processors, and assemblers.

Other structures at the airport include a nine-story hotel and a new terminal complex. An enclosed shopping mall and other commercial facilities are situated adjacent to the airport industrial park. The airport also has 90,000 square feet of warehouse space used for cargo operations and storage.

2.1.8 Aesthetics

Military structures currently located on Air National Guard land at the BIA consist of large airplane hangars and one and two-story administrative and maintenance buildings. These are interspersed with

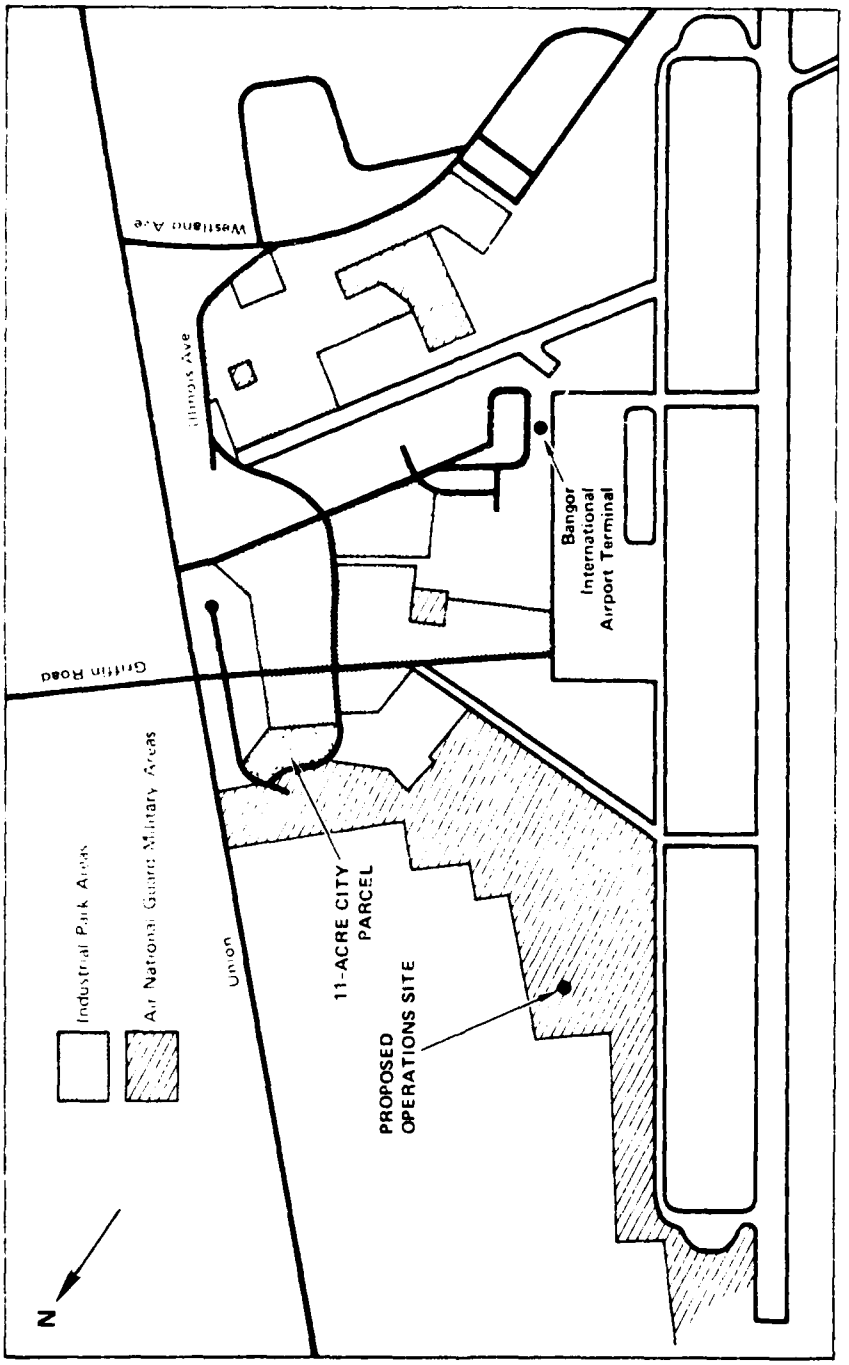

FIGURE 2-3 BANGOR INTERNATIONAL AIRPORT AREA

undeveloped open space and some wooded areas. The proposed site is visible from other areas of the BIA, including the industrial park.

2.2 Affected Biophysical Environment--Bangor

2.2.1 Physiography

The terrain at the BIA is gently rolling with an average slope of 3 percent and a maximum slope of 10 percent. The BIA is about 130 feet above sea level.

2.2.2 Ecology

Bangor lies within the Laurentian Mixed Forest Province ecoregion (Bailey, 1976). Local vegetation is that typical of northern hardwood-spruce forests (Kuchler, 1975). In such areas, either mixed coniferous/deciduous stands or a mosaic-like pattern of pure coniferous and pure deciduous stands occur. Tree species present include maple, beech, yellow birch, hemlock, elm, basswood, and white pine. Eighty percent of Penobscot County is forested.

In developed portions of the military area on which the operations center would be sited, tree cover has been removed and replaced with indigenous grasses. On the northwest edge of the site, there are secondary growth trees and underbrush consisting of such hardwood species as oak and elm and scrub. Most of the land at the site of the proposed operations center has been cleared and graded.

The ranges of a number of species of fauna and flora with endangered or rare status include all or part of the State of Maine. Bald eagles have been sighted in the vicinity of Bangor. However, the BIA does not provide critical habitat for the eagle or any other endangered species (Perry, 1981; Kale, 1981).

2.2.3 Soil

Bangor is in the U.S. Department of Agriculture's designated Northeastern Forage and Forest region and within the land resource area known as the New England and Eastern New York Upland. Soils are generally dense and brittle. However, the soil erodibility factor in that area is in the median range, between highly susceptible and barely susceptible (Department of the Interior, 1972).

In general, the proposed site of the operations center is covered by 6 to 8 inches of top soil underlain by clay and sandy clay. Solid rock is close to the surface, and outcroppings occur in the northwest corner of the Air National Guard parcel.

The soil is exposed to an average of 37 inches of rain and 94 inches of snow each year.

2.2.4 Water

Bangor has an ample supply of water. The Bangor Water District has water rights to sources that could potentially supply 15-16 million gallons per day (gpd). Currently used sources have a capacity of 8 million gpd (Caldwell, 1981). In addition, the Bangor Water District has attained a high rating for the quality of its water (Greater Bangor Chamber of Commerce, 1978).

The Bangor water system serves roughly 50,000 people and industrial and commercial customers in the region. Water deliveries average 5.5 million gpd or 75 percent of the system's capacity (Caldwell, 1981). The water district supplies freshwater to the BIA through two 16-inch mains. Surface water in the Kenduskeag Stream that flows near the proposed operations site is classified as adequate for support of plant and animal life and for human recreation, but inadequate for human consumption.

Groundwater can be found at the OTH-B operations site at depths from 1 to 8 feet below grade. These depths are typical of groundwater conditions in the Bangor area.

Wastewater from the Air National Guard area is treated by the City of Bangor's primary treatment facility, in which flows average 3.5 to 4.0 million gpd in very dry periods and in which peak flows have reached 18 million gpd in extremely wet periods. Peak flows are so great because the city storm drain system is interconnected with the sewerage system (Hambrock, 1981). The facility currently serves a resident population of approximately 32,000 plus the commercial and industrial firms in Bangor. The Bangor Pollution Abatement Facility was designed to serve an anticipated population of 43,000 by the year 1985; however, it has space for additional treatment units to service 50,000 persons. The facility can now accommodate a maximum of 35 million gpd (during wet flow periods) for primary treatment; it will be expanded to a maximum capacity of 43 million gpd, and activated sludge (secondary) treatment is planned for the mid-1980s. The Air National Guard parcel at the BIA received new sewer lines in the mid 1970s.

BIA has a well-developed surface water drainage system. A major drain runs parallel to Kenduskeag Stream, and no drainage problems have occurred on the Air National Guard parcel.

2.2.5 Air

Bangor is located within the State of Maine's Downeast Air Quality Region. Air quality in the municipality of Bangor has been designated nonattainment (i.e., in excess of federal standards) for total suspended particulates (TSP) and carbon monoxide (CO). Urban fugitive dust (from sanding of paved streets, construction activities, and storage areas) is the principal "nontraditional" source of particulates in the Bangor

area. The traditional source categories of TSP are industrial fuel combustion, commercial/institutional fuel combustion, residential fuel combustion, and solid waste disposal (Maine Department of Environmental Protection, 1979). High carbon monoxide levels in the central Bangor area are the result of vehicles (Garvey, 1980).

A State Implementation Plan (SIP) for improving air quality in Bangor was approved by the Maine Board of Environmental Protection in May 1979. It has also been approved by the federal Environmental Protection Agency (EPA). As part of the SIP, current strategies to bring the Bangor area into attainment for TSP include: road cleaning; dust suppression during construction; street sweeping, vacuuming, and flushing; paving of parking lots; and adding curbs to paved roads. To improve CO levels, the state has instituted the Federal Motor Vehicle Emissions Control Program and will enforce the law that prohibits tampering with pollution control equipment on vehicles. In addition, traffic signals will be improved and traffic patterns will be modified. These strategies appear to be effective in reducing pollutant levels (Emory, 1981; Wood, 1981).

Data provided by a TSP monitor on top of an Air National Guard building at BIA indicate that TSP emissions do not exceed standards in the vicinity of the airport. The federal secondary standard for TSP is 150 micrograms per cubic meter and the federal primary standard is 260 micrograms per cubic meter. Monitoring indicates that maximum TSP levels at the BIA were 92 micrograms per cubic meter in 1980. In March 1981, the average was 99 micrograms per cubic meter (Emory, 1981). Although levels of TSP are increasing--as the airport is developed, construction occurs, and ground vehicle traffic becomes more common--they do not approach the maximum levels set by the standards. Aircraft take-offs and landings do not currently result in significant TSP emissions; the total contribution of air traffic to ambient TSP levels is estimated at 5 micrograms per cubic meter per day. CO levels at the airport are also within the standards. Consequently, the BIA is now considered a clean air area (Emory, 1981).

2.2.6 Noise

Because of the proximity of the proposed operations facility to the airport, potential noise contributions from constructing and operating the facility are a concern. Preliminary information concerning 1980 noise levels indicates that in the northeast portion of the Bangor ANG Base the day-night sound level (DNL) exceeds 65 dBA, whereas in the southwest portion of the Base nearest the main airport runway, DNL exceeds 75 dBA (Hoyle, Tanner, & Associates, 1980).* At the site of the

* Information on existing and projected noise at the Bangor Air National Guard (ANG) Base is obtained from a preliminary draft of the Comprehensive Master Plan Update for the Bangor International Airport. This information is subject to revision upon completion of the master plan.

proposed operations facility, existing noise levels range from DNL 70 dBA to DNL 75 dBA. (DNL of 70 dBA and 75 dBA are approximately equal to L_{10}* of 73 dBA and 78 dBA, respectively). The Federal Highway Administration's (FHWA) exterior noise standards for nonresidential structures specify that L_{10} should not exceed 75 dBA.

Available noise information does not reflect the effects of localized intermittent phenomena. A jet engine test facility is located immediately south of the site. Engines are tested at the facility approximately 20 times a year. The duration of each test is approximately 68 minutes. Noise monitoring data for operation of this facility are not available; however, it is clear that operation of this facility would contribute to increasing the identified noise levels at the site. The preliminary information available to date indicates that the noise levels at the site will exceed the L_{10} 75 dBA standard when the jets are being tested.

The U.S. Department of Housing and Urban Development (HUD) has developed exterior standards that classify areas where DNL exceeds 65 dBA as "discretionarily unacceptable." For the interiors of residences, DNL 55 dBA is the maximum recommended noise level. Preliminary noise information for 1980 indicates that DNL exceeds 65 dBA in the southeastern portion of the existing housing owned by the Air Force.

Available preliminary information indicates that noise levels at the operation site will be lower in the future than they were in 1980. This expectation may be attributable to anticipated aircraft fleet modifications involving engine replacement. Future noise levels will be within the range of DNL 65 dBA to DNL 70 dBA or approximately L_{10} 68 dBA to 73 dBA. These estimates do not consider the effects of noise from the jet engines test facility. Because data on the facility's contribution to noise levels are unavailable, a worst-case scenario is assumed for this supplement. Even so, FHWA noise standards of L_{10} 75 dBA (exterior) would be exceeded only intermittently, on certain occasions when the jet engine test facility is in use. Preliminary estimates of future noise at the Air Force housing on Union Street indicates noise levels below DNL 65 dBA. On this basis the area would be classified as acceptable according to HUD standards.

2.3 Somerset County

The setting near the proposed transmitter site in Somerset County is substantially unchanged from the description in the 1975 EIS. The site is still surrounded by forest products company land, remote from all permanent habitation. This context is significant in the evaluation

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* A sound level that is exceeded 10 percent of the time is represented by the symbol L_{10}.

and update of health and safety related effects--the biological effects of nonionizing radiation, safe separation distances for electroexplosive devices (EEDs), and electromagnetic interference with cardiac pacemakers.

Section 3

UPDATE OF THE ENVIRONMENTAL CONSEQUENCES OF THE PROPOSED ACTION

3.1 Bangor Region of Influence

3.1.1 Socioeconomic Consequences

The socioeconomic effects of the action need to be identified because they may cause biophysical impacts via increased land development, traffic, or other such change. The driving force behind the estimation of socioeconomic impacts is the expenditure to construct and operate the operations center at BIA. These expenditures would in turn result in additional economic activity in the Bangor ROI. This section estimates the extent and size of these additional or secondary effects and their potential effects on the biophysical environment.

The basic data used to estimate impacts are construction expenditures and employment. In addition to the integrated operations, maintenance, and security facility, the Air Force plans to construct a commissary, base exchange, dining hall, dormitory, gymnasium, and other support facilities, and to refurbish existing base facilities. Total construction expenditures have been estimated at $15.4 million in 1983 and $5.6 million in 1984. The employment estimates for the 1983 to 1986 period are shown in Table 3-1.

Table 3-1

AIR FORCE AND CIVILIAN PERSONNEL ESTIMATES
FOR BIA OPERATIONS CENTER

	Year			
	1983	1984*	1985	1986
Military				
Officers	7	21	42	42
Enlisted	3	217	435	435
Total	10	238	477	477
Air Force civilians		16	32	32
Civilian contractor employees	30	35	15	-
Total	40	289	524	509

* Military personnel and Air Force civilians estimated at 50 percent of 1985 staffing.

3.1.1.1 Employment

An increase in income in the Bangor ROI would produce increases in employment if there were no excess capacity in the trade and services sectors. On the basis of an estimated average salary of $12,000 (Hoyle, Tanner, and Associates, 1981, adjusted for inflation), employment increases are estimated as shown in Table 3-2.

Table 3-2

ESTIMATED EMPLOYMENT CHANGES IN BANGOR ROI

	1983	1984	1985	1986
Direct	40	289	524	509
Secondary including construction	815	550	485	445
	855	839	1009	954

The maximum percentage impact on secondary employment would occur in 1983, when construction activity would be highest. If all construction employees live in the Bangor ROI, the temporary increase in employment could reach 815, or 2 percent of the 1983 labor force of approximately 41,000. Over the long term (1986 and beyond) the operation center could result in the creation of approximately 445 secondary jobs, or 1.1 percent of the labor force, implying a reduction in the unemployment rate of 1.1 percent.

3.1.1.2 Population

It is estimated that in 1983 the total project work force would be 855 persons, 730 of whom would be construction workers. It is assumed that much of the construction force would be drawn from the Bangor-Brewer ROI and surrounding areas. These workers would commute from their present residences on a daily basis. The balance of the construction force would be transient workers who would reside in motels and other temporary lodging or in campers.

Because the influx of Air Force personnel during operation is likely to exceed the number of construction workers coming from outside the ROI and because the Air Force workers would be located in the region for an extended period of time, the primary population impacts would result from the Air Force employees and their dependents. In 1985, the Air Force operational work force would consist of 509 persons--477 military personnel and 32 civilians. If the average family size in the military is the same as the Air Force-wide average, the population increase from military personnel in 1985 would consist of 811 dependents in addition to the 477 military personnel, for a total of 1,288. If all

32 civilian personnel were hired from outside the ROI, they and an additional 55 dependents could enter the ROI. The total change would then be 1,375 persons, or 1.7 percent of the projected 1985 population.

The influx of Air Force personnel would not be expected to cause secondary population growth in the Bangor-Brewer area. In 1980, there were 2,860 unemployed persons (7 percent of 40,800) in the region. Given the extent of unemployment, the 445 secondary jobs created over the long term would probably be filled by the resident ROI labor force or by dependents of personnel working at the operations center. Therefore, any employment induced by the presence of the Air Force is likely to be absorbed by the present labor force and not cause secondary population growth.

Between 1970 and 1980, the Bangor ROI population grew at an average annual rate of 0.3 percent. With the proposed action, the total annual average growth rate would be approximately 0.7 percent between 1980 and 1985.

Both the anticipated rate of population growth and the absolute amount of growth are small and unlikely to cause significant adverse impacts. Subsequent analyses address the ability of particular elements of the infrastructure (e.g., housing, educational facilities) to accommodate the expected increase.

3.1.1.3 Income

Changes in income would result directly from the salaries of incoming contractor and Air Force personnel and indirectly from the spending for personal items, procurement, and military construction. The total change in personal income was calculated and is shown in Table 3-3. The maximum impact will occur in 1985, when income increments would total approximately $13.2 million, for an increase of 1.8 percent over income levels expected with the no-action alternative. (The maximum income impact does not coincide with the maximum impact on secondary employment because construction employment does not generate as much secondary income as does military employment.) The increase in per capita income would be less than $100.

3.1.1.4 Housing

Between 1983 and 1985, 509 Air Force households would seek housing in the Bangor ROI. Military housing would consist of 165 existing single family units and 140 dormitory beds in a facility to be constructed, and thus could absorb the demand for 305 units. The balance, 204 households, are expected to seek housing in the region.

It is estimated that there were approximately 1,200 vacant units in the ROI in 1979. It has also been estimated that non-Air Force demand for housing between 1980 and 1985 would result in the construction of an

Table 3-3

DIRECT AND INDIRECT CHANGES
IN PERSONAL INCOME IN THE BANGOR REGION OF INFLUENCE
(Millions of 1980 Dollars)

	1983		1984		1985		1986	
Direct		1.1		4.5		7.4		7.0
Indirect								
Personal consumption	1.0		3.4		5.1		4.7	
Procurement	-		-		0.7		0.7	
Construction	8.8		3.2		--		--	
Total indirect		9.8		6.6		5.8		5.4
Total		10.9		11.1		13.2		12.4

additional 600 to 3000 units. It is reasonable to expect that the housing stock would be sufficient to accommodate Air Force generated demand.

3.1.1.5 Educational Facilities

The average number of school age children per Air Force worker is 0.46 for military and 0.71 for civilians (SRI International and Air Force Engineering Services Center, 1979). Therefore, the action could bring about 240 new pupils to the ROI.

Without the proposed action, the population in the ROI is estimated to increase to 81,000 in 1985. If the ratio of student population to total population remains at the 1980 level the number of pupils would increase by approximately 100 students to 13,800. In the ROI student enrollments have declined and several schools have been closed over recent years. In Bangor, the 14th Street elementary school is being held in reserve should enrollments increase in the future. Consequently, the educational staffing and facilities should be sufficient to accommodate the influx of Air Force school children.

3.1.1.6 Community Facilities and Services

Because the population changes likely to result from the proposed project are small, no noticeable impacts are anticipated.

3.1.1.7 Land Use

The construction of the OTH-B radar operations center and support facilities at BIA constitutes a continuation of military use of the land

in that area. There are a number of similar commercial structures on airport land and in the near vicinity at the present time. No impact on land use is expected.

3.1.1.8 Aesthetics

The proposed facilities would be similar to the one-story commercial and military structures currently located at BIA. Therefore, no modification in the appearance of the BIA region is likely.

3.1.2 Biophysical Consequences

3.1.2.1 Physiography

Most of the land on this parcel has been cleared and graded. Additional grading would be required for site preparation, however, significant disruption of the terrain and drainage patterns would not result.

3.1.2.2 Ecology

The construction and operation of the OTH-B radar operations center at BIA would not have a notable impact on plant or animal species in the area. The proposed site of the facility is partially cleared, and tree cover has already been removed. The location does not provide a habitat for any rare, endangered, or threatened species (Perry, 1981; Kale, 1981).

3.1.2.3 Soil

It is unlikely that any soil erosion problems would result from construction of the operations center facilities at BIA (Kale, 1981).

3.1.2.4 Water

Adequate water supplies are expected to be available for the operations center and AF domestic uses. The demand of the operations center and Air Force residences would be far less than the estimated present reserve capacity of 2.5 million gpd. The distribution system is also adequate to accommodate new construction at BIA (Caldwell, 1981).

The 1,375 additional residents of the Bangor ROI in 1985 would use 151,000 gpd of water. This projection is based on an estimated daily consumption per person of 110 gallons derived from current water demand and current population data. The incremental increase in water demand is approximately 6 percent of the city water supply system's reserve capacity of 2.5 million gpd.

The Bangor sewerage system can also accommodate the output of the operations center. The operations center could be easily connected to the existing sewer lines that serve the airport (Hambrock, 1981).

The new population would also generate a total of approximately 165,000 gpd of sewage to be handled by the city sewerage system. The city sewage treatment plant has sufficient reserve capacity--more than 5 million gpd during wet periods--to handle the incremental demand.

A drainage ditch for surface runoff at the airport has sufficient capacity to handle the increased runoff that would be caused by development of the proposed site.

3.1.2.5 Air

The construction and operation of the OTH-B radar operations center at BIA is not expected to cause air emissions great enough to exceed the standards specified in the Maine State Implementation Plan. However, fugitive dust will be generated during construction, and it may be necessary to implement certain dust control measures to reduce particulates.

During operation, emissions will be generated by routine, biweekly, 4-hour testing of the back-up diesel generator and by employee vehicle traffic. The diesel generator is assumed to be between 500 and 1,000 kW in size. Routine testing would result in much lower emissions than would result from operating the generator on a continuous basis, as it would be during a power failure. The TSP and CO emissions caused by testing or temporary operation of the generator are likely to be less than those generated by one aircraft landing-take-off cycle at the airport (Air Force, 1980). During infrequent planned exercises the generator might emit about 8 pounds per day of particulates and 30 pounds per day of CO (SRI, 1980). In comparison, a jumbo jet landing-take-off cycle would generate approximately 1.5 pounds of particulates and 47 pounds of CO (Golden, 1979). To reduce the impact of the generator on air quality, the Air Force plans to use high quality diesel fuel and to install the best available pollution control devices (Air Force, 1980).

Traffic generated by the 509 civilian and military personnel working at the operations center would have a minimal effect on local air quality. The center's operation would probably add fewer than 800 cars per day to the existing volume of airport traffic.

All new residents associated with the proposed facility would reside within the Downeast Air Quality Region. In 1980, total population in the four counties (i.e., Hancock, Washington, Penobscot, and Piscataquis) that comprise the Air Quality Region was estimated at 227,000; thus the new population of 1,375 would be an increase of approximately 0.6 percent in the region as a whole. Incremental emissions attributable to the new population would also be small. On the basis of current levels of emissions (from all area and point sources except industrial), current population data, and annual emission factors, the increases in TSP, CO, SO_x, NO_x, and hydrocarbons would

probably be less than 1 percent of all emissions in the region in that year (EPA, 1978). Because the new population will probably locate throughout the ROI and not just in Bangor, related emissions are likely to be generated outside the central Bangor area.

3.1.2.6 Noise

Some noise would be caused by construction activities in the immediate area of the operations center; however, other construction activities are being conducted at the airport, and all construction noise would be inconsequential by comparison with the noise of aircraft traffic.

Any noise created by operation of the diesel generation equipment or the employee vehicle traffic associated with the OTH-B radar operations center would also be minimal compared with the noise generated by aircraft traffic at the BIA.

3.2 Electromagnetic Interference (EMI) Consequences--Somerset County

The reduction in transmitted power density described briefly in Section 1.1.2 and in detail in Appendix A will in general reduce the distance from the transmitter site in Somerset County at which EMI affects any given system by 50 percent for airborne systems (see section A.3.2, p. A-5) and by approximately 30 percent for ground-level systems (see Section A.3.3, p. A-7). Because the safe separation distances for electroexplosive devices (EEDs) and for artificial heart pacemakers relate to potentially significant hazards to personnel and depend significantly on power densities very close to the transmitter, calculations for those systems are updated in Appendix B.

No significant risk to cardiac pacemaker wearers is now expected at electric field strengths less than 200 V/m (equivalent to 10 mW/cm^2 power density), in contrast with the 50 V/m susceptibility limit that was used in the 1975 EIS. This 4-fold improvement in shielding from electric fields corresponds to a 16-fold increase in power density that current pacemaker units have been measured to withstand. Moreover, pacemakers are less susceptible at the frequencies used by the OTH-B radar than at higher frequencies, and are less susceptible to the frequency modulation used by the OTH-B than to amplitude modulation, particularly the pulse modulation used by most radars. Because of both decreased susceptibility and decreased transmitter power, no hazard to wearers of cardiac pacemakers is now expected outside the site exclusion fences, in contrast with the 4,400-ft separation distance in the 1975 EIS. The 200 V/m exposure boundary, shown in Figure B-1, p. B-3, is well within the exclusion fence for each sector transmitter.

Although the susceptibility limits for EEDs have not changed, the planned power density reduction significantly reduces the safe separation distances. The maximum distance for safe separation is

reduced from 22,000 ft (1975 EIS) to 14,000 ft for the most sensitive class of EEDs. Hazard area boundaries for EEDs and other detailed information on these devices is given in Appendix B.

3.3 Human Health Consequences--Somerset County

3.3.1 Conclusions

Radiation safety is a vital consideration. In recognition of the increased public awareness of and controversy over biological effects of RFR, a critical review and analysis of the scientific literature in this field was performed and is presented in Appendix C. That information supersedes all related information in the 1975 EIS, in particular that in Appendices A, B, and C. Studies of bioeffects at frequencies up to about 18 GHz were reviewed, and those judged most significant scientifically were selected for analysis from the large body of available literature.

The calculations presented in Appendix A and affirmed by measurements on the ERS indicate that the power density of radiofrequency radiation (RFR) in nearby communities due to operation of the OTH-B transmitter will be less than 0.00001 mW/cm^2, and that near the site exclusion fences the power density will be less than 1.1 mW/cm^2. Everywhere along the exclusion fence the power density will be below the more stringent safety standard proposed by the American National Standards Institute Committee C95.4 to replace the current U.S. standard (see pp. C-8 and C-9).

No scientific evidence was found to indicate that any ill effects will result from exposure to the RFR from the OTH-B radar. This conclusion is based on 1) an assessment of the scientific literature, 2) a comparison with current and proposed standards, and 3) the remote location of the transmitter site. The relatively few retrospective epidemiology studies of health effects from RFR exposure done in the United States and the USSR are not considered to contain evidence that the RFR from the OTH-B radar will constitute a hazard to the population. This judgement is supported by the findings of the many investigations conducted in the laboratory with experimental animals, tissues, cultures, and microorganisms. A review of the major findings of Appendix C is presented below.

3.3.2 Review of Major Findings

In the first few sections of Appendix C, the sources of bioeffects literature and the criteria used for selecting documents for review are discussed, and the problems of risk assessment are treated. Recent results of measurements of environmental levels of RFR in major U.S. cities, performed by the Environmental Protection Agency, are presented. The current exposure standards of various countries are compared, and proposed revisions of the U.S. standard are considered.

In Section C.5, the interactions of RFR with biological entities are discussed, leading to the concept of"specific absorption rate" (SAR) of RFR energy, and we describe how the SAR of a biological entity varies with its size, shape, orientation, and constituents, and with the frequency, polarization, and power density of the incident RFR. An important finding is that there are frequencies at which the SARs for the whole body and for major extremities (head, limbs) are highest, denoted as "resonant" values. Knowledge of such values for various species (or models thereof) permits scaling of approximately equivalent dose rates from one species to another (with varying degrees of validity because of biological differences among species). Also briefly discussed in this section is the often encountered confusion and controversy over "thermal" versus "nonthermal" RFR bioeffects.

The present state of knowledge about the bioeffects of RFR is discussed in detail in Section C.6. Representative retrospective epidemiologic studies conducted in the United States, Poland, Czechoslovakia, and the Soviet Union are analyzed. Such studies are directed toward determining whether one or more health-related conditions can be associated statistically with purported or actual exposure of humans to RFR. Included in this section are studies of RFR-induced eye damage (cataractogenesis) in humans. Although the validity of retrospective epidemiologic studies is difficult to assess because of the lack of adequate exposure information and because of the influence of uncontrolled factors, such studies do provide almost the only direct information on the possible effects of RFR on humans. The U.S., Polish, and Czechoslovakian epidemiologic studies analyzed offer no clear evidence of detrimental effects of chronic exposure of the general population to the levels of RFR assumed. Regarding RFR cataractogenesis, isolated cases of eye damage have been reported for occupationally exposed individuals, but the epidemiologic studies provide no convincing evidence of cataractogenesis from cumulative exposure to low levels of RFR. In contrast, the Soviet findings, which are consistent with the voluminous, earlier Soviet literature, suggest that occupational exposure to RFR at average power densities less than 10 mW/cm^2 does result in various symptoms, particularly those associated with disorders of the central nervous system. Because the USSR symptomatology has never been reported in Western studies and because of the marked differences between Soviet and Western publications in the procedures used for reporting data, any prediction of possible RFR hazards based on the USSR epidemiological studies would require acceptance of these Soviet findings at face value. Thus, there is no reliable epidemiological evidence that chronic exposure of humans to the levels of RFR from the OTH-B radar outside the exclusion fence will be hazardous to their health.

Over the past 30 years, studies have been conducted on possible mutagenic and cytogenetic effects of RFR on rats, mice, fruit flies, and microorganisms. Also, at least one published report has suggested that chronic exposure to RFR might induce cancer. No evidence has been found

that exposure to RFR induces mutations in bacteria or fruit flies. Belief that it induces mutations in mammals requires acceptance at face value of two studies that are demonstrably badly designed and executed and have numerous flaws in the reporting, analysis, and intepretation of the data. Evidence for cytogenetic effects of RFR is mixed. However, the lowest power density at which cytogenetic effects were reported was 20 mW/cm^2, a value considerably higher than the levels from the OTH-B radar outside the exclusion fence. There is no evidence that chronic exposure to RFR causes induction of any form of cancer, even at power densities of the order of 100 mW/cm^2.

"Teratogenesis" usually refers to the induction of anatomical aberrations in a developing fetus by a physical or chemical agent. However, teratologic studies have also included observation of fetal death and/or resorption and of physiological and cellular abnormalities in the offspring observed postpartum. In addition, the effects of RFR on the development of eggs of birds and pupae of the darkling beetle have been studied. The results support the conclusion that such effects are due to the heat produced by the RFR rather than from any special teratogenic properties of RFR. Thus, there is no evidence that the RFR from the OTH-B radar beyond the exclusion fence is potentially hazardous to prenatal children at any stage of development.

Extensive studies of ocular damage in animals from exposure to RFR have been conducted. The results from such studies indicate that RFR cataractogenesis is essentially a gross thermal effect that has a threshold power density at which the difference between the rates of heat generation by RFR and heat removal is large enough to result in damage to the lens of the eye. The mean threshold values probably vary to some extent from species to species, but are of the order of 100 mW/cm^2. Thus, chronic exposure to the RFR from the OTH-B radar beyond the exclusion fence is most unlikely to result in eye damage because the power densities there are far below the threshold.

Several types of studies have been conducted on effects of RFR on the nervous system of animals. These studies are considered particularly important in the USSR, where RFR is believed to stimulate the nervous system directly and thereby cause a variety of physiological effects. Scientists in the United States tend to doubt that RFR interacts directly with the nervous system, except possibly under special circumstances, and they consider most effects of RFR on the nervous system to be indirect results of other physiological interactions. The exceptions refer to effects ascribed to RFR characteristics that are not relevant to the OTH-B radar, such as pulsed and amplitude-modulated RFR, and include the RFR hearing effect and the calcium efflux phenomenon.

The existence of a "blood-brain barrier" (BBB) in most regions of the brain has been established experimentally. This barrier normally provides high resistance to movements of large molecules (e.g., proteins

or polypeptides) from the blood vessels into the surrounding brain tissue, presumably to protect the brain from invasion by various blood-borne pathogens and toxic substances. Several investigators have reported that low levels of RFR can increase the permeability of the BBB to certain substances of large molecular weight. However, others were unable to confirm such effects, and the subject remains controversial. Recent findings indicate that hyperthermic levels of RFR can alter the permeability of the BBB. It is also possible that exposure at average power densities of the order of 10 mW/cm^2 may result in randomly distributed, clinically subacute, reversible alterations. However, additional research using current or improved methodology is necessary to ascertain whether chronic exposure to nonhyperthermic levels of RFR affects the BBB. At present, there is no evidence that alterations of the permeability of the BBB would result from exposure to the levels of RFR from the OTH-B radar outside the exclusion fence.

Experimental results indicate that RFR can cause observable histopathologic changes in the central nervous system (CNS) of animals, but it appears that these changes are thermal in nature. There is no evidence that such changes would be caused by exposure to the RFR from the OTH-B radar outside the exclusion fence.

Many studies have been conducted on apparent alterations of the electroencephalogram (EEG) and/or evoked responses (ERs) of animals exposed to RFR. Some of these have been performed with metal electrodes either implanted in the brain or attached to the scalp during exposure. Such metallic electrodes grossly perturb the fields and produce greatly enhanced absorption of energy (i.e., field enhancement) in the vicinity of the electrodes. Such increases probably result in highly localized transient heating in the immediate vicinity of conductive implants in tissue exposed to microwave fields with time-averaged intensities greater than 1 $microwatt/cm^2$. The greatly enhanced fields themselves are also likely to cause artifacts in nervous system tissue function in the volume immediately around the electrode because of the sensitivity of such tissue to electrical stimulation. Such artifacts may be minimized by use of electrodes appropriately designed from high resistivity materials. Experiments in which such specially constructed electrodes were used, or in which electrodes were applied after exposure, show no evidence of statistically significant differences in EEGs or evoked responses between control and RFR-exposed animals.

A very large number and variety of behavioral studies in animals exposed to RFR have been conducted, including studies of effects on reflex activity, RFR-perception studies, evaluations of effects of RFR on learning and on performance of trained tasks, studies of interactive effects of RFR and drugs on behavior, and investigations of behavioral thermoregulation. Some of these investigations seem to have originated from studies in the USSR claiming that RFR had direct effects on the CNS at low power densities. Evidence to support this claim from neurophysiological studies in the United States is meager, and the

behavioral evidence also does not generally support the claim. The studies on RFR as a noxious stimulus do not show that the animals can perceive RFR as such. Radiation avoidance has been observed, but appears to be part of the thermoregulatory behavior of animals; when the environment is cold, animals will use RFR as a source of warmth. Disruption of performance or learning appears to have rather high power-density thresholds. Interaction of RFR with drugs affecting the CNS appears to be the most sensitive behavioral response to RFR, but even these studies do not prove a direct effect of RFR on the CNS. Overall, the behavioral studies do not indicate a special effect of RFR on the nervous system, and the mechanism of most of the results remains conjectural. These studies provide no evidence that exposure of humans solely to the RFR from the OTH-B radar outside the exclusion fence will alter their behavior. There is some evidence for synergistic effects on rats of certain drugs and RFR at average power densities (for 2.45 and 2.8 GHz) of the order of 1 mW/cm^2, the approximate level at one point of the exclusion fence. However, there is no evidence that such synergistic effects would occur in humans in the nearby communities, where the average power densities are much lower.

Exposure of animals to RFR has produced somewhat inconsistent effects on the endocrine system of mammals. In general, the effects produced appear to be related to either the heat load associated with the RFR or the stress induced in the animals by the RFR and, possibly, other experimental circumstances such as handling. Some effects also appear to be related to alteration of the circadian rhythm by RFR. There do not appear to be any effects clearly demonstrated to be associated with nonthermogenic stimulation of the endocrine system or the associated parts of the CNS. Because the reported effects of RFR on the endocrine systems of animals are largely ascribable to increased thermal burdens, stresses engendered by the experimental situation, or both, there is no evidence that such effects would occur in humans exposed to the RFR from the OTH-B radar at the power densities outside the exclusion fence.

RFR does appear to have effects on the immune system of mammals. Some of the reported effects were obtained at low power-density levels, but most of the studies were performed at relatively high power densities, and in some cases effects obtained at high power densities were not found at lower power densities, suggesting the possibility that power-density thresholds exist. Some of the results indicate immunosuppressive effects, some indicate immunostimulative effects, and some indicate that the state of the immune system depends on the duration of exposure or when measurements were taken relative to the time of exposure or the time of day.

Effects on the immune system from chronic exposure to RFR at low power densities (tens to hundreds of $microwatts/cm^2$) are unlikely to be linked simply to temperature increases. The existing evidence indicates that some of the immune-system effects are probably mediated

through the effect of RFR on the endocrine system, involving the general syndrome of adaptation to stress. The mechanisms and significance of such effects are not yet understood, nor have individual findings been independently verified. There is currently no evidence that reported RFR effects on the immune systems of animals at average power densities less than 1 mW/cm^2 would occur in humans exposed to the RFR from the OTH-B radar outside the exclusion fence, or that such effects would be hazardous to human health. If chronic low-level RFR exposure did impair the ability to resist disease, then a relatively high rate of infectious diseases should occur among people occupationally exposed to RFR, and it is highly unlikely that this would not have been noticed.

The literature on biochemical and physiological effects associated with RFR is extensive. Many of the reported effects are associated with other events (e.g., changes in hormonal levels or stress adaptation), some are questionable for various reasons, and others do not have a clear medical significance. The thermal basis for reported *in vivo* biochemical and physiological effects of RFR is evident. Most significant are the investigations with nonhuman primates because of their close anatomical and physiological similarities to humans, and especially relevant to the OTH-B radar are the results of exposures of rhesus monkeys at frequencies in the HF range. These results showed that exposures to average power densities exceeding 100 mW/cm^2 were within the thermoregulatory capabilities of these animals. Also noteworthy were the negative results of the blood-chemistry assays performed on rhesus monkeys 1 to 2 years after such high-power-density exposures. The investigations with other species exposed at higher frequencies (148, 918, and 2450 MHz) and lower power densities yielded a variety of positive and negative results. However, none of the results indicates that exposure of humans to the levels of RFR from the OTH-B radar outside the exclusion fence will induce any detectable biochemical or physiological effects.

In overall summary, the basic conclusion of the 1975 EIS is unchanged: no reliable evidence has been found to indicate that any hazard will result from either short-term or prolonged exposure of people to the power densities from the OTH-B radar transmitter outside the exclusion fence. However, certain gaps remain in our knowledge of the biological effects of RFR. These gaps may be identified as follows:

(1) Available results from animal studies and mathematical models are insufficient to permit adequate predictions regarding similar biological effects in humans. Moreover, most animal research has not involved continuous exposure over periods approaching an animal's lifetime. These deficiencies affect the interpretation of experiments done at different RFR frequencies and the interpretation of differences in biological response among various species.

(2) Prospective epidemiological studies of effects of exposure of humans to RFR are lacking. Existing epidemiological studies, while extensive and reasonably well-done, are all retrospective in nature, and are therefore subject to inherent defects of method.

The probability that new information would reveal a significant hazard cannot be dismissed, but is judged to be relatively low.

3.3.3 Other Viewpoints

Some of the general concerns that have been expressed by others regarding the assessment of the biological effects of RFR are: first, there are insufficient data upon which to base an assessment of potential hazard to human health; second, research on the effects of long-term, low-level exposures is only in its infancy; third, little is currently known about the details of mechanisms of interaction of RFR with biological tissues, with the consequence that potentially hazardous effects that may occur have not been more precisely targeted for study; fourth, there are specific studies in the literature that report effects at average power densities less than 100 microwatts/cm^2; fifth, even though some studies report negative findings (i.e., no effects as a result of RFR exposure), such negative findings can possibly be attributed to faulty experimental design or procedures; sixth, epidemiological studies from the Soviet Union have reported various symptoms in persons exposed for many years to RFR at levels in the range from tens to hundreds of microwatts/cm^2--symptoms that when taken together are called the "microwave radiation syndrome"--but that such symptoms are not recognized in Western epidemiology studies; seventh, although we know a lot more today than we did 10 years ago, we will know even more 10 years from now and it is therefore likely that with this additional knowledge will come recognition of new, hazardous effects of long-term, low-level exposure to RFR; eighth, safe power thresholds for RFR exposure of the general population have not been established, and, further, safety standards vary from country to country; and ninth, there has been insufficient research on possible alterations of genetic material and carcinogenic effects of long-term, low-level exposure to RFR.

Documentary evidence that has been cited as reason for these concerns includes: the studies on calcium efflux changes; the studies on blood-brain barrier permeability changes and modifications of behavior; the studies on changes in the immune system; and the studies on changes in permeability of the blood-brain barrier to certain radiotracer-labelled molecules. Many of these studies are discussed in Appendix C.

Section 4

UPDATE OF THE AFFECTED ENVIRONMENT AND ENVIRONMENTAL CONSEQUENCES OF THE ALTERNATIVES

This section describes the affected environment and environmental consequences of (1) the alternative of not building the operations center, and (2) the alternative of locating the maintenance and security personnel for the receiver site at Bucks Harbor, and locating the maintenance and security personnel for the operations center and transmitter site with the operations personnel at Bangor.

No Action Alternative

If the system is not deployed, the socioeconomic and biophysical consequences in Bangor and Bucks Harbor would not occur. The ERS would be dismantled and removed and the OTH-B program would be terminated. The benefits to the security of the nation from operating the OTH-B radar would not accrue.

Bucks Harbor Alternative

In addition to changes in the proposed action, the Air Force has identified a new alternative which calls for a permanent maintenance and security force of 144 persons to be stationed at Bucks Harbor AFS to serve the Columbia Falls receiver facilities. If this alternative is implemented, 6 officers, 131 enlisted, and 7 civilian Air Force personnel would be assigned.

For the Bucks Harbor alternative, construction expenditures would be required to provide housing, new support facilities, and to repair existing buildings. Construction expenditures would total $5.9 million in 1983 and $2.1 million in 1984.

This alternative would allow a reduction in the staffing at BIA compared to the proposed action. Under the Bucks Harbor alternative, staffing at the BIA operations center would be 40 officers, 353 enlisted, and 20 civilians for a total of 413 positions. However, the total staffing would be 48 positions greater than for the proposed action because of the need to maintain two locations for the staff.

The impacts in the Bangor ROI should be similar in nature but of lesser magnitude than those of the proposed action since the staffing at BIA is 20 percent less than for the proposed action. The impacts of the alternative in the Bangor ROI are characterized by the impacts described in Section 3 for the proposed action, and are not discussed further.

4.1 Affected Environment--Bucks Harbor

4.1.1 Affected Socioeconomic Environment

Bucks Harbor Air Force Station is located approximately 8 miles south of the town of Machiasport on the west shore of Machias Bay. Bucks Harbor AFS is in south-central Washington County, the easternmost county in Maine. This analysis of socioeconomic conditions and potential impacts focuses on communities in southern Washington County. The Bucks Harbor ROI is depicted in Figure 4-1. The communities included are: Addison, Cherryfield, Columbia, Columbia Falls, Cutler, East Machias, Harrington, Jonesboro, Jonesport, Machias, Marshfield, Milbridge, Rogue Bluffs, Steuben, Whiting, and Whitneyville.

Bucks Harbor AFS is a 90-acre facility and would provide some housing and support facilities. Current Air Force missions at Bucks Harbor are being phased out in 1981. The majority of Air Force and civilian personnel assigned to the proposed security and maintenance force are expected to reside at Bucks Harbor or in the nearby ROI communities.

4.1.1.1 Employment

The Bucks Harbor ROI had a labor force of 6,576 in 1980 (Maine Department of Manpower Affairs, 1981b). The unemployment rate averaged 10.8 percent in 1980 as compared with 7.7 percent in the state and 7.1 percent in the United States. Based on the projected population and on the 1980 labor participation rate (0.44) the labor force in 1985 would include about 7,300 people.

4.1.1.2 Population

The population of the 17 communities in the Bucks Harbor ROI totaled approximately 14,800 in 1980, a 23 percent increase from the 1970 population (See Table 4-1). Growth trends varied considerably among the communities. The growth in the towns with populations greater than 1,000 in 1970 (i.e. East Machias, Jonesport, Machias, and Milbridge) were less than the ROI average.

If the population of the ROI continues to grow at the rate it did between 1970 and 1980 the population would be approximtely 16,400 in 1985 (an increase of about 1,600).

4.1.1.3 Income

The total personal income in the ROI in 1980 is estimated to be $95.9 million (1980 dollars). Based on projected population and historic growth in real income the total personal income in 1985 will be $117.0 million (1980 dollars). These figures were derived from the 1979 income in Washington County (Gilcreast, 1981).

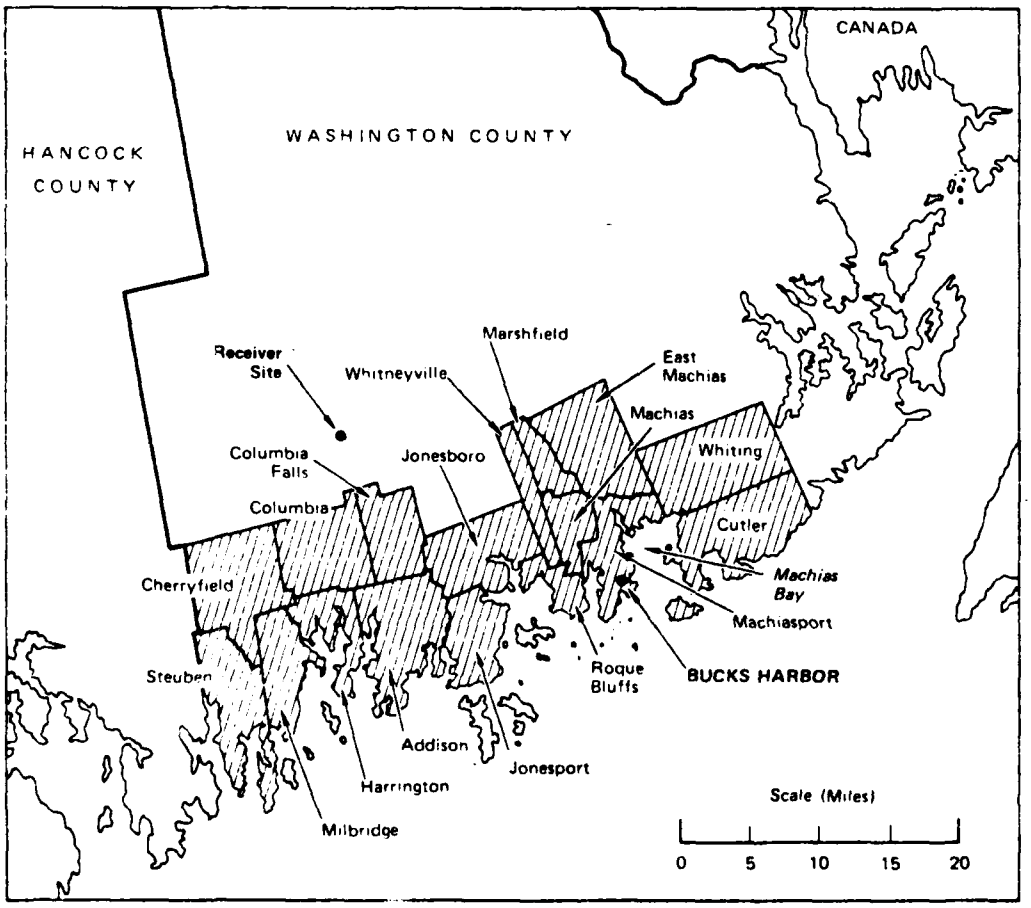

FIGURE 4-1 BUCKS HARBOR REGION OF INFLUENCE

Table 4-1

POPULATION IN THE BUCKS HARBOR ROI

	1970	1980	Percent Change
Addison	773	1,060	37
Cherryfield	771	986	28
Columbia	162	276	70
Columbia Falls	367	516	41
Cutler	588	728	24
East Machias	1,057	1,235	17
Harrington	553	860	56
Jonesboro	448	552	23
Jonesport	1,326	1,509	14
Machias	2,441	2,460	1
Machiasport	887	1,110	25
Marshfield	227	417	84
Milbridge	1,154	1,303	13
Rogue Bluffs	153	241	58
Steuben	697	962	38
Whiting	269	331	24
Whitneyville	155	264	70
	12,028	14,810	23

Source: Department of Commerce, Bureau of the Census, 1980.

The estimated per capita income in the ROI was $6,478 in 1980 and $7,120 in 1985. This compares with the statewide average of approximately $8,100 in 1980.

4.1.1.4 Housing

According to the 1980 census, there are 7,080 housing units in the Bucks Harbor ROI. This represents a 32 percent increase over the 1970 figure. The increases can be attributed to a 23 percent growth in the population and a decrease in the number of persons per housing unit (2.24 in 1970 and 2.09 in 1980). For Maine as a whole, the average number of persons per housing unit was 2.24 in 1980.

If the population in the ROI is 16,400 in 1985 and the average household size remains constant (2.09) by 1985 the housing stock would increase by 767 units to approximately 7850. Existing military housing consists of 27 family units and 45 beds for unaccompanied personnel.

4.1.1.5 Educational Facilities

The number of pupils residing in the Bucks Harbor ROI was 3,032 in the 1980-1981 school year (Fish, 1981). The ratio of student population to total population is 0.20. Despite the increase in total population, school enrollments have been decreasing. It is estimated that between the 1979 and 1980 school years the number of pupils in the ROI declined by approximately 70.

4.1.1.6 Community Facilities and Services

Publicly provided facilities and services in the ROI are adequate for the population in the region.

4.1.1.7 Land Use

Land use in the region surrounding Bucks Harbor is primarily residential, agricultural, and recreational. A small percentage of land is used for commercial uses (mostly timbering and fish processing).

Bucks Harbor Air Force Station consists of 90 acres of land, 50 acres of which are 5 interconnected parcels and the rest of which are easements and rights-of-way. All present AFS activities take place outside the coastal zone regulated by state law. The AFS contains a radar facility that will be phased out by the end of 1981, an FAA facility, and several unused buildings.

4.1.1.8 Aesthetics

The AFS is situated on a hill and is visible from Highway 92. Radar and radio towers are located on Howard and Miller Mountains, both approximately 900 feet above sea level.

4.1.2 Affected Biophysical Environment

4.1.2.1 Physiography

Bucks Harbor AFS is situated on the rocky coast of Maine which consists primarily of ledges and shelves, although these are interspersed with some level areas suitable for building. Most of the facilities of Bucks Harbor AFS are located on top of a steeply inclined hill about 250 feet above sea level. Slopes on the site range from less than 3 percent to over 30 percent.

4.1.2.2 Ecology

Bucks Harbor AFS lies within the Laurentian Mixed Forest Province ecoregion (Bailey, 1976). Typical vegetation is that of a northeastern spruce-fir forest (Kuchler, 1975). Such forests usually occur on flat plains and are characterized by northern white cedar, tamarack, maple, birch, eastern hemlock, and eastern white pine. Forested areas cover 80 percent of Washington County. There are some wooded areas consisting of hardwood and scrub pine on the Station.

This vegetation provides habitat for a variety of animal species including the endangered bald eagle. There are wintering and nesting eagles in the area, including several pair on the islands in Machias Bay, however, none are known to winter or nest on Bucks Harbor AFS. Other wintering birds also use the islands in Machias Bay, however, none has been observed using the habitat on the Air Force Station (Spencer, 1981 and Todd, 1981). Animals that have been spotted within Bucks Harbor AFS include deer and porcupine.

4.1.2.3 Soil

The U.S. Department of Agriculture has classified the southern half of Maine as having low susceptibility to erosion based on slope and rainfall factors. The coast however, is more suceptible to erosion than inland areas (Department of the Interior, 1972). Erosion is a potential problem in certain areas of Bucks Harbor AFS (Bahr, 1981). Average annual snowfall is 60 inches; normal annual rainfall is 44 inches.

4.1.2.4 Water

Currently, water supplies at Bucks Harbor are derived from two groundwater wells, and there is not a scarcity of supply. One well has an estimated capacity of 70 gallons per minute (Bahr, 1981).

On the Air Force Station, there is a secondary treatment facility with more than sufficient capacity to handle sewage from the residences and small industrial operations there. The treatment facility has been permitted by the State to accommodate up to 40,000 gallons per day. Septic systems are commonly used for sewage disposal outside the Station.

4.1.2.5 Air

Bucks Harbor AFS lies within the Downeast Air Quality Region. Although one area of Washington County has been designated nonattainment for secondary TSP standards, the area around Bucks Harbor is considered to have pristine air. The only major emission sources in the vicinity that have been granted air permits are several fish processing facilities (Emory, 1981).

4.1.2.5 Noise

There are no major sources of noise on Bucks Harbor AFS.

4.2 Environmental Consequences--Bucks Harbor

4.2.1 Socioeconomic Consequences

4.2.1.1 Employment

The income change in the Bucks Harbor ROI would increase employment if there is no underutilized labor in the trade and services sectors. The direct and maximum potential secondary employment increases are shown in Table 4-2.

Table 4-2

ESTIMATED EMPLOYMENT CHANGES IN BUCKS HARBOR ROI

	1983	1984	1985
Direct	-	72	144
Secondary	280	130	61
Total	280	202	205

The employment changes for 1983 and 1984 are primarily caused by construction of support facilities and do not include effects from construction of the receiver facility.

The maximum percentage impact on employment would occur in 1983 when construction activity would be highest. If all construction employees live in the Bucks Harbor ROI, then the impact on employment could reach 280 or 4.0 percent of the labor force in 1983. This employment change could lead to a reduction of the local unemployment rate to around 7 percent from its 1980 level of 10.8 percent. Over the long term (1985 and beyond) the Bucks Harbor alternative could result in the creation of approximately 60 secondary jobs or less than 1 percent of the ROI labor force.

4.2.1.2 Population

During the construction phase, it is expected that most workers will be drawn from the ROI and would not cause any additions to the population of the ROI. Those workers who come from outside the ROI are likely to take up temporary residence and leave when work is completed. The possible influx of construction workers is less than seasonal variations caused by tourists and summer residents.

In 1985 the maintenance and security force at Bucks Harbor would consist of 144 persons--137 military and 7 civilians. If all Air Force employees came from outside the region, the total population increase would be approximately 390 persons. This amounts to a 2.4 percent increase over the projected 1985 population of 16,400 in the ROI. The population growth rate between 1980 and 1985 would average 2.6 percent per year compared to 2.1 percent for the 1970 to 1980 decade.

The influx of Air Force personnel would not be expected to cause secondary population growth in the Bucks Harbor ROI. There are projected to be approximately 790 unemployed persons in the ROI in 1985. Therefore, the 60 secondary jobs created by the Bucks Harbor alternative can easily be filled from the ranks of the unemployed.

The anticipated rate of population growth and the absolute amount of growth are nominal and do not indicate that adverse impacts are likely. Subsequent analyses address the ability of particular elements of the infrastructure to accommodate the estimated changes.

4.2.1.3 Income

The potential income changes result directly from the salaries of incoming Air Force personnel and indirectly from the spending for personal items, procurement, and construction. The estimates are summarized in Table 4-3. The long term increase in personal income is estimated to be $2.7 million per year in the Bucks Harbor ROI. This would be approximately 2.3 percent over the projected personal income of $117 million for 1985. Per capita personal income in the ROI would be virtually unchanged by the alternative action.

Table 4-3

DIRECT AND INDIRECT CHANGES IN PERSONAL INCOME
IN BUCKS HARBOR ROI
(millions of 1980 dollars)

	1983		1984		1985	
Direct		-		0.9		1.9
Indirect from						
Personal consumption			0.3		0.7	
Procurement					0.1	
Construction	3.4		1.2		--	
Total Indirect		3.4		1.5		0.8
Total		3.4		2.4		2.7

4.2.1.4 Housing

Between 1983 and 1985, 144 Air Force households would seek housing in the Bucks Harbor ROI. Military housing would consist of 27 existing single family units, 30 family units to be constructed, and 45 existing beds for unaccompanied personnel, and thus absorb the demand for 102 units. The remaining 42 households are expected to seek housing in the ROI.

The housing stock in 1985 is projected to be 7580 units. The long term demand for 42 units could be filled by 1 percent of this housing stock.

4.2.1.5 Educational Facilities

The action would increase student enrollment in the ROI by approximately 70 students by 1985. Because of declining enrollments (70 in 1980) there should be sufficient capacity to accommodate Air Force students.

4.2.1.6 Community Facilities and Services

Because the action would result in a small increase in population, adverse impacts are not anticipated.

4.2.1.7 Land Use

The proposed action represents a continuation of military use on Bucks Harbor AFS. The Station is located on a hill above the coast; therefore any activity would be situated outside the coastal zone area that is regulated by state law.

Fewer than 50 acres would probably be required for use by the new population. Use of the environmentally sensitive areas in the immediate coastal zone would be controlled by the Maine Department of Environmental Protection.

4.2.1.8 Aesthetics

The proposed action would not change the aesthetic character of the Station. The structures would be visible from several locations on the base but would be similar in design to structures currently present.

4.2.2 Biophysical Consequences

4.2.2.1 Physiography

The construction of operations facilities and 30 housing units would not result in significant disruption of the terrain and drainage patterns.

4.2.2.2 Ecology

There are no known plants or animals inhabiting Bucks Harbor AFS that would be adversely affected by the construction and operation of a maintenance and security facility there. Local wildlife experts believe that the eagles on the islands in Machias Bay would continue their nesting activities unjeopardized by the proposed activity (Todd, 1981 and Spencer, 1981).

The 390 new residents that would be associated with the OTH-B facilities at Bucks Harbor Air Force Station would probably restrict their activities, for the most part, to the mainland. As long as they do not interfere directly with the eagle and waterfowl nesting areas on the islands in Machias Bay, their presence would not jeopardize the birds.

4.2.2.3 Soils

Potential soil erosion problems may result from construction of the facility at Bucks Harbor as the soil in the area is fine grained, consists of clay, and is silty on slopes (Kale, 1981 and Bahr, 1981). However, standard engineering practices would be taken to prevent erosion during and after construction.

4.2.2.4 Water

Water supplies from wells are expected to be adequate on the AFS and the existing primary treatment plant could be used to service the proposed facilities (Bahr, 1981). The sewage plant on the Station is equipped to treat waste generated by over 200 persons.

New residents would require an additional 39,000 gallons of water a day, based on estimated consumption of 100 gallons per person per day (Salvato, 1972). Local groundwater supplies are adequate to accommodate the new population.

Based on an estimate of 90 gallons per person per day, the new population would generate approximately 35,000 gallons per day of sewage. The existing primary sewage treatment facility at Bucks Harbor AFS with a capacity of 40,000 gpd could accommodate those living in Bucks Harbor AFS housing. However the facility would have to be modified for secondary treatment to comply with federal standards. Those personnel living off base would utilize present sewerage and septic tank systems in the ROI.

4.2.2.5 Air

The only effect on air quality created by the proposed facilities and activities would be the traffic generated by the OTH-B system personnel (144 military personnel and dependents). Air quality should not be degraded significantly by fewer than 150 cars arriving and departing the AFS daily. Because Bucks Harbor is on the coast, offshore winds would often act to disperse pollutants emitted by these mobile sources.

Incremental pollutant emissions created by the new population (0.2 percent of the current regional population) in the vicinity of Bucks Harbor would likely represent an additional 0.2 percent of total emissions in the Downeast Air Quality Region as a whole. The impact would be greater in the ROI and in the vicinity of Bucks Harbor and Columbia Falls, however, the increase in emissions is not expected to cause local air quality levels to change noticeably.

4.2.2.6 Noise

The only noise resulting from the proposed action would be that associated with the increase in automobile traffic and temporary construction activities.

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Appendix A

POWER DENSITY CALCULATIONS

A.1 Introduction

The information in this appendix supersedes that given in paragraph 1.e(4), p. 13, and in Appendix A of the EIS. Moreover, because this revision was prompted by a planned reduction in the maximum power density of the transmitter system, all environmental consequences that depend on power density are also revised. The sections of the EIS affected by these revisions are listed in Appendices B and C of this supplement.

Power density information is presented for two cases of interest: along the axis of maximum power density of the mainlobe, and along the ground directly below that axis. Depending on transmission frequency, the axis is elevated approximately 12 to 22 degrees, and the mainlobe extends approximately ± 3 degrees in azimuth and ± 8 to ± 16 degrees in elevation about the axis. Electromagnetic radiation near the axis comprises the so-called space or sky wave. It is relatively unaffected by the properties of the earth in the vicinity of the transmitting antenna. Radiation along the ground below the axis comprises the so-called surface or ground wave. The ground wave is strongly affected by the earth. The power density at intermediate elevations is not discussed here, but it would be a complicated mixture of the two wave types. From the power density of the mainlobe and the relationship of sidelobes to the mainlobe, power density information is derived for sidelobes both in front of and behind the backscreen (backlobes) of the antenna array, for both space and ground waves.

This appendix describes power densities and other characteristics for a single 60-degree sector. Expansion of the installation from one to three sectors for full 180-degree coverage implies that the total power density at any given location will be the sum of the power densities from each operating sector.

These power density calculations have been affirmed by measurements taken at the ERS transmitter site for the four bands installed there. Calculations of power densitites from the ERS are adjusted for the smaller groundscreen of the ERS, and compared with measurements on the ERS in Section A.6, p. A-13.

A.2 System Parameters

Original and revised system parameters are given in Table A-1. The gain of the antenna is expressed both as a ratio and in common engineering notation (decibels). The combined effect of reducing the

Table A-1

CHANGES IN THE OTH-B RADAR TRANSMITTER PARAMETERS

System Characteristics	Original	Revised
Continuous wave power (MW)	2.4	1.2
Antenna gain[a], mainlobe maximum		
Ratio	320	160
Decibels	25	22
Antenna gain, sidelobe maximum[b]		
Ratio	16	8
Decibels	12	9
Antenna gain, backlobe maximum[b]		
Ratio	3.2	1.6
Decibels	5	2

[a] All gain figures are stated relative to an isotropic distribution.

[b] The term "sidelobe" is used here to refer to antenna lobes in front of the backscreen other than the mainlobe, whereas "backlobe" denotes any antenna lobe radiating behind the backscreen.

transmitter power by 50% and the antenna gain by 50% is to reduce the maximum power density to 25% of the value originally planned. Coincidentally, the azimuthal width of the mainlobe is doubled. As before, the maximum power density for sidelobes shown in Table A-1 is a factor of 20 smaller than the maximum power density in the mainlobe, and the backlobes (antenna lobes radiating behind the backscreen) have a maximum power density a factor of 100 smaller than the mainlobe maximum.

A.3 Calculating Maximum Power Densities

A.3.1 Introduction and Assumptions

It is impractical to design a single antenna array to accomplish the objectives of the OTH-B radar throughout the entire band of frequencies needed (5-30.6 MHz). Consequently, six separate antenna arrays are planned, only one of which will be driven at any given time. Each array consists of 12 elements, uniformly spaced in a plane in front of a vertical conducting backscreen. The six arrays are aligned over a common groundscreen as shown in Figure A-1. Each array serves the specific band of frequencies shown in Table A-2. Both the groundscreen and backscreen are constructed of galvanized wire mesh.

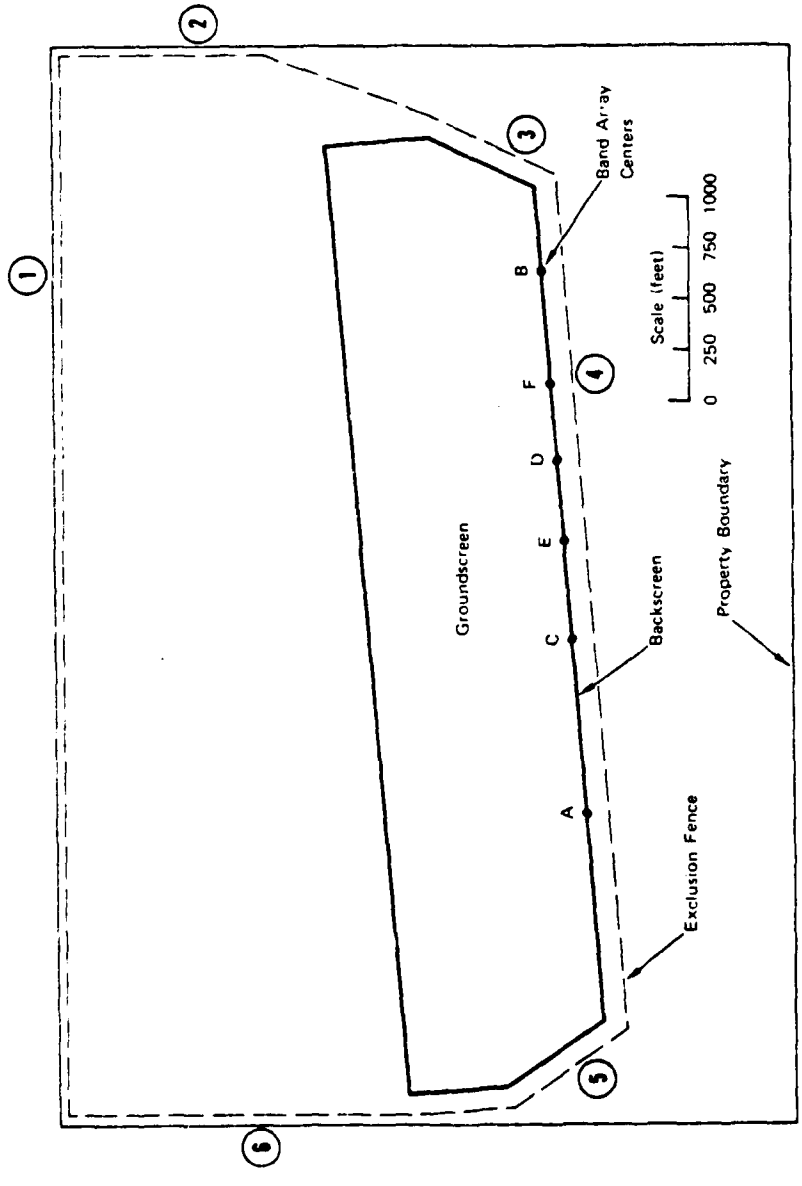


FIGURE A-1 SITE DIAGRAM

Table A-2

ANTENNA ARRAY CHARACTERISTICS

Band	Frequency(f), MHz	Array Length (D), ft	Nearfield Extent ($0.1\ D^2/L$), ft	Farfield Start ($0.5\ D^2/L$), ft
A	5.00-6.74	1016	610	3050
B	6.74-9.09	754	450	2250
C	9.09-12.25	559	335	1675
D	12.25-16.50	415	250	1250
E	16.50-22.25	308	185	925
F	22.25-30.60	226	135	675

Because of the array configuration, the OTH-B radar will produce a radiation field that is concentrated in a fan-shaped beam, narrow in azimuth, much wider in elevation. The azimuth of the mainlobe axis is steered $\pm$30 deg from the direction perpendicular to the backscreen by varying the phase (timing) of the signals fed to the 12 elements of the array that is in use. The mathematical description of the complete field produced by antenna arrays is very complicated. Therefore, approximate expressions have been developed to facilitate calculation.

For an idealized antenna in free space, the resulting electromagnetic field is normally described by dividing it into three regions, to which different sets of analytical conditions apply. Those regions are the nearfield, the transition region, and the farfield. The boundaries of the three regions are not sharply defined; rather, field conditions gradually change with increasing distance from the center of the antenna. Different approximations apply to the different regions. The boundaries between the regions are defined by the acceptable error in the approximations that are made.

The farfield is defined as a region over which the analytic conditions are constant and the fields vary inversely with distance (i.e., the power density varies inversely with the square of the distance). The distance from the array center beyond which the true farfield exists is $2D^2/L$, where D is the array length and L is the radiation wavelength (equal to the speed of light divided by the frequency). At distances between $0.5\ D^2/L$ and $2D^2/L$, the field conditions resemble those in the farfield region, but the sidelobes

increase slightly in strength, and pattern nulls are somewhat filled in. For convenience and because it involves no significant errors, we henceforth treat this region as part of the farfield. The nearfield exists in the region between the array center and 0.1 D^2/L. Here the sidelobes are not identifiable, and the on-axis field strength varies rapidly in a complex manner. Between 0.1 D^2/L and 0.5 D^2/L, there is a transition region where the pattern is irregular and the sidelobes degenerate into bumps on the sides of the mainlobe. With some care in interpretation, those idealized concepts can be applied to arrays of the type used in the OTH-B radar.

For the mainlobe of antenna arrays such as the OTH-B radar, idealized as a rectangular aperture, the maximum power density values in free space for distances out to 2 D^2/L are shown in Figure A-2. Beyond a distance equivalent to 0.5 D^2/L, the variation in power density is substantially the same as for the farfield, varying as the reciprocal of the distance squared. Between the distance 0.1 D^2/L and 0.5 D^2/L the actual power density begins to vary rapidly at amplitudes not exceeding the line shown in Figure A-2, which varies as the reciprocal of the distance (Hansen, 1964). In the nearfield, at distances out to 0.1 D^2/L, the power density continues to vary rapidly in a complex manner, but because the finite extent of the source is dominant in this region, the power density does not exceed the line shown in Figure A-2, which varies as the reciprocal of the square root of the distance (Hansen, 1964). Consequently, the use of the maximum values shown in Figure A-2 serves to adequately bound the expected power densities on and near the axis of the mainlobe. Table A-2 gives the extent of the nearfield and transition region for each of the six arrays. The wavelength used for each band corresponds to a frequency near the midpoint for that band; this provides an entirely adequate approximation, given that the bands are relatively narrow and the field boundaries are not sharply defined.

A.3.2 Free Space Power Densities

At any specific distance R from the antenna, the power density in free space is given by the following expressions, in which the coefficients are adjusted to conform to our usage of power density in milliwatts/cm^2, and the symbols have the following meanings:

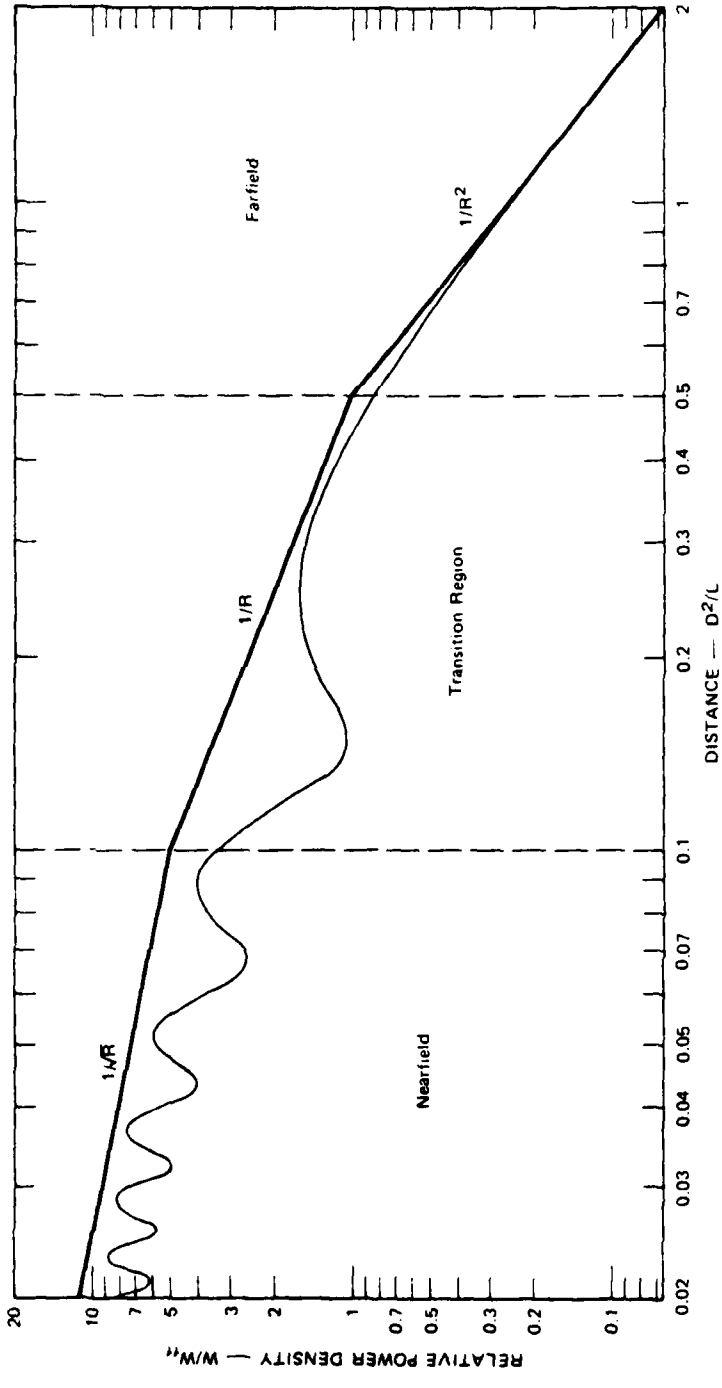
P = total transmitted power, continuous wave (watts)

G = antenna gain (ratio)

D = array length (ft)

R = distance from array midpoint (ft)

L = radiation wavelength (ft).



NOTE: Heavy Line Represents Values Used in Three Regions

FIGURE A-2 MAINLOBE ON-AXIS POWER DENSITY FOR A RECTANGULAR ANTENNA

For R greater than $0.5\ D^2/L$ (farfield), the power density W (mW/cm^2) is:

$$W = \frac{0.086\ PG}{R^2} \quad .$$

Hence, the on-axis power density in the mainlobe is:

$$W = \frac{1.6 \times 10^7}{R^2} \quad .$$

The symbol W_{ff} is used to denote the power density at the start of the farfield for a particular band, and is obtained from the above expression by using the corresponding values given in Table A-2. Then the on-axis power density in the transition region (see Figure A-2) is:

$$W = \frac{(W_{ff})(0.5D^2/L)}{R}$$

and the on-axis power density in the nearfield (see Table A-2) is:

$$W = \frac{(5W_{ff})(0.1\ D^2/L)^{1/2}}{R^{1/2}}$$

The maximum power density encountered outside the mainlobe and forward of the backscreen in free space can be obtained from the corresponding mainlobe expressions by reducing the calculated power density by a factor of 20, and for maximum power densities behind the backscreen by a factor of 100 (see Table A-1).

A.3.3 Ground-Level Power Densities

Because the ground is not a perfect conductor, considerable attenuation of the free space power density occurs near ground level. For most soil conditions and over most distances of interest (farfield), the effect of ground attenuation is to introduce another factor $1/R^2$ into the power density expressions (Norton, 1936). However, for distances in the near field and transition regions the attenuation varies approximately as $1/R^n$, where n varies with frequency and soil conditions. For damp terrain (least attenuation), n ranges from 0.20 for band A to 0.66 for band F in the near field, and from 0.63 to 1.29 respectively for the transition region (Norton, 1937a, 1941). Only the free space spreading loss, as described by the expressions in Section A.3.2, is used for the range within 1,000 ft of the antenna, because the earth is covered by the highly conductive groundscreen.

Ground-level power densities are further affected by the orientation of the dipole array elements. Bands E and F use the

vertical orientation, for which the attenuation characteristics in the preceding paragraph apply. However, the elements of the other bands are oriented at a 45 deg angle to the vertical. In effect, half the radiated power can be considered to be radiated with vertical polarization and half with horizontal polarization. The horizontal component is effectively suppressed at ground level because of the conducting earth (Norton, 1937b, 1941). Hence, for bands A, B, C, and D, the ground-level power density is further reduced by 50% at all distances of interest.

With these considerations and the free space power density expressions, the maximum power densities at ground level can be calculated. The results are presented in Figures A-3, A-4, and A-5. For distances in the farfield beyond those shown in the figures, the coefficients in Table A-3 may be used to obtain power densities with the following expression:

$$W = \frac{(\text{Table A-3 coefficient})(10^{10})}{d^4} \quad \text{mW/cm}^2$$

where d is the distance in feet along the ground from the array midpoint. The left and right sidelobes differ in power density because the effective groundscreen length differs with band placement along the array.

A.4 Maximum Power Densities at the Exclusion Fence

From the results in Section A.3.3, the maximum ground-level power density at each of six sites of interest denoted on Figure A-1 can be calculated. The results are listed in Table A-4. Of all points outside the exclusion fence exposed to a ground-level mainlobe maximum, site 1, the point nearest to the band B array, yields the largest power density. Corresponding sites for the other bands (all along the far fence approximately 2600 ft. from the backscreen) yield values in the range of 0.3 to 0.6 mW/cm2.

Sites 2 and 6 are the points nearest an array that could be considered to be exposed to a portion of the [illegible]iation field that may have power densities as great as values on [illegible] axis of the mainlobe. Hence, the values shown in Table A-4 f[illegible] [illegible]ces were not reduced by the sidelobe factor, although points farther out on the same azimuth, in the farfield, would be. This procedure yields 1.1 mW/cm2 for site 2, the highest value for any site along the exclusion fence.

Site 4, closest to the band F backlobes, yields 0.6 mW/cm2, the highest value for the area behind the backscreen. The calculated power densities at sites 3 and 5 are much smaller than those for the comparable sites 2 and 6 because, although closer to the arrays, they must be considered to be reduced by the sidelobe factor.

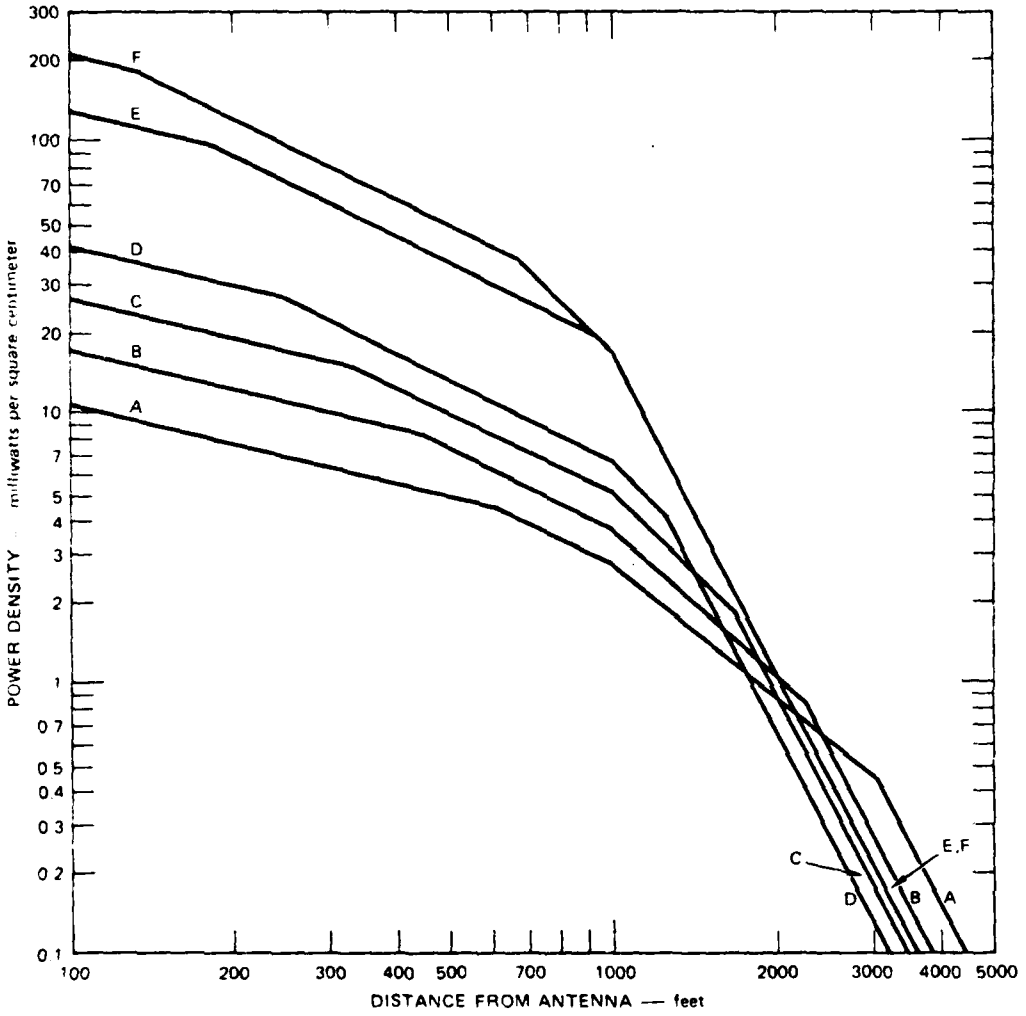


FIGURE A-3 CALCULATED POWER DENSITIES AT GROUND LEVEL FOR MAINLOBE

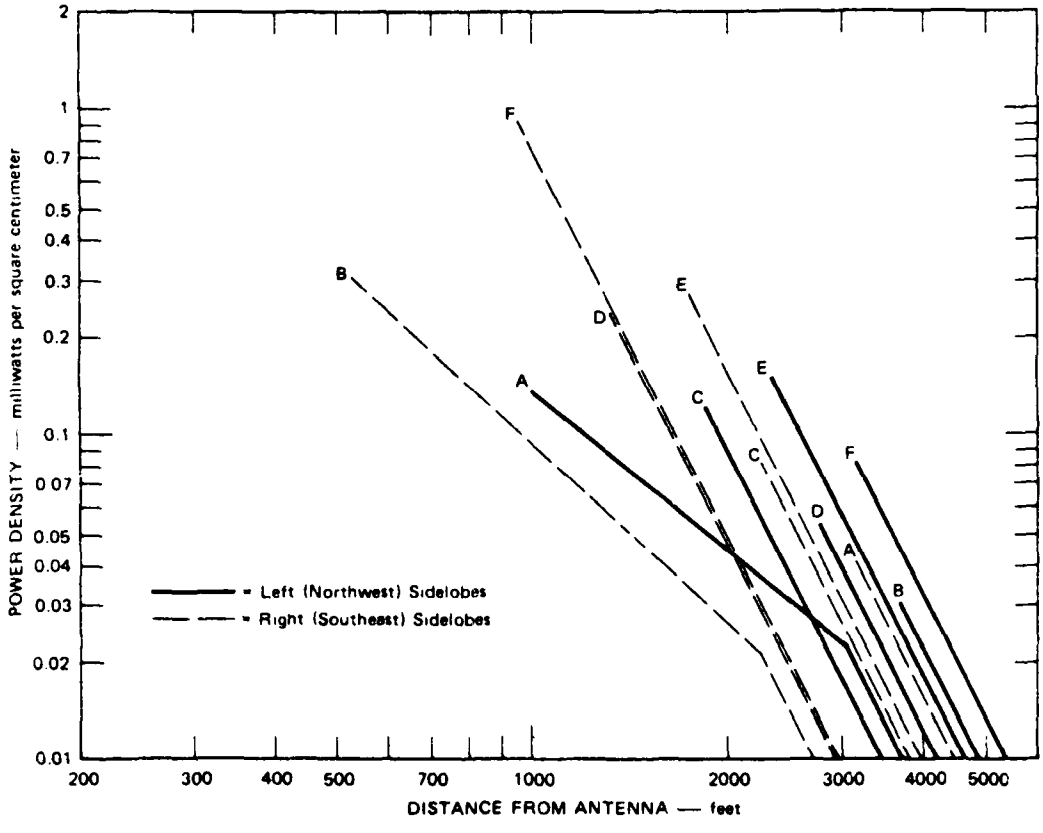


FIGURE A-4 CALCULATED POWER DENSITIES AT GROUND LEVEL FOR SIDELOBES

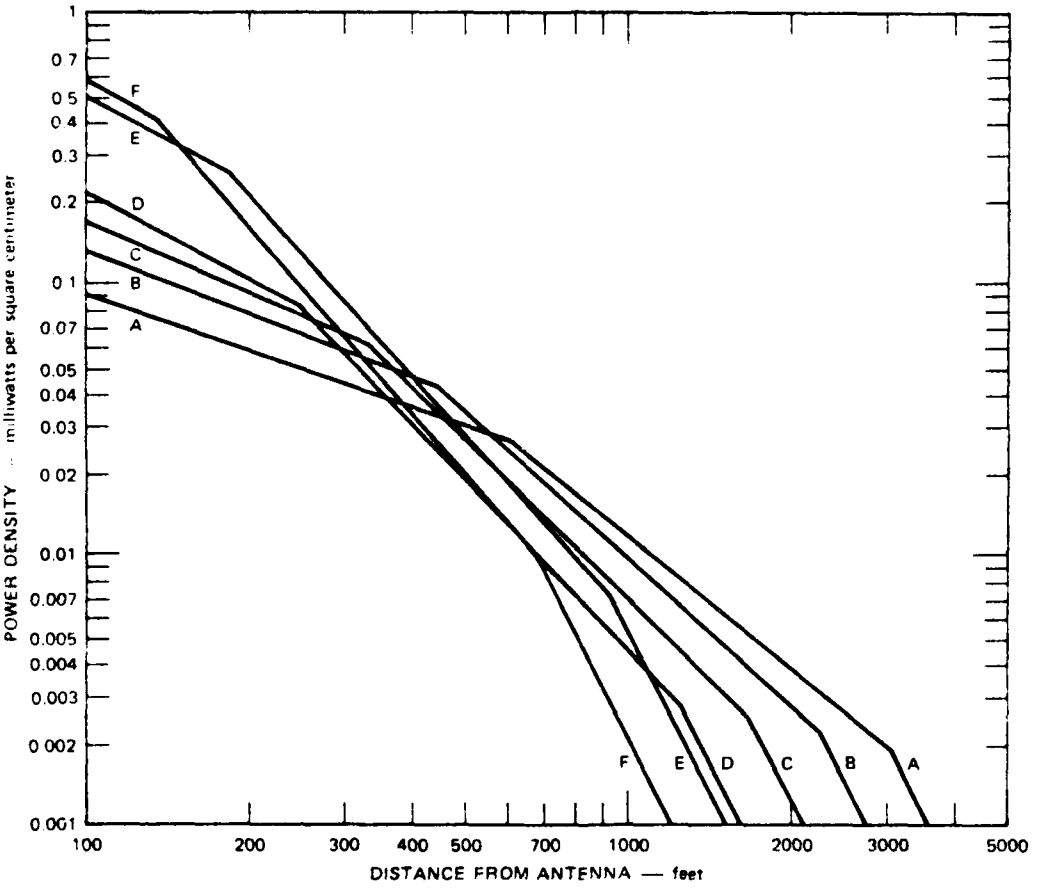


FIGURE A-5 CALCULATED POWER DENSITIES AT GROUND LEVEL FOR BACKLOBES

Table A-3

COEFFICIENTS FOR FARFIELD GROUND LEVEL POWER DENSITIES

Band	Coefficients			
	Mainlobe	Left Sidelobe[a]	Right Sidelobe[a]	Backlobe
A	3,700.	190.	390.	16.
B	2,100.	550.	54.	5.6
C	1,400.	140.	210.	1.9
D	990.	310	72.	0.67
E	1,600	450.	250.	0.52
F	1,600	800.	74.	0.21

[a] Left sidelobes radiate to the northwest, and right sidelobes to the southeast, in the first sector to be constructed (replacing the ERS).

Table A-4

MAXIMUM CALCULATED POWER DENSITIES AT THE EXCLUSION FENCE

Site Number	Band	Frequency(f), MHz	Ground-Level Power Density, mW/cm^2
1	B	6.74-9.09	0.7
2	B	6.74-9.09	1.1
3	B	6.74-9.09	0.3
4	F	22.25-30.60	0.6
5	A	5.00-6.74	0.1
6	A	5.00-6.74	0.8

An alternative view of power densities near the exclusion fence is presented in Figure A-6. A composite boundary formed of the limits for each band proposed by the ANSI subcommittee (see Section C.2.4, p. C-7, and Figure C-1, p. C-9) is shown along with a 1 mW/cm^2 contour for reference.

Because of the rapid falloff in power density near the antennas, the contribution from the other two sectors to the power density within or near the exclusion fence of a sector would be negligible.

A.5 Electric Field Intensities

For assessing hazards to artificial heart pacemakers, the electric field intensity corresponding to a given power density is needed. The electric field intensity E, given in V/m, may be calculated from:

$$E = (3770\ W)1/2$$

where W is the power density in mW/cm^2.

A.6 Power Density Measurements at the ERS Transmitter Site

A.6.' Background

A measurement team visited the Experimental Radar System (ERS) transmitter site on 9 and 10 June 1981 to make measurements of the power density at selected points in the vicinity. Since the ERS transmitter operates below the 1.2-MW power level of the eventual OTH-B system, the measurements were then scaled up to indicate the levels that would have existed if the transmitter power had been 1.2 MW.

The measurement locations are indicated by numbers 1 through 14 on Figure A-7. Positions 1 through 4 are about 10-15 ft inside the exclusi fence in front of the antenna array centers for bands C, E, D, and B, ectively. Positions 5 through 8 are all 5-10 ft outside the exclusion fence and alongside the present access road. Position 9 is about 20 ft outside the fence and 3 ft in front of the plane of the backscreen; it is therefore behind the array's dipole elements. Position 10 is the same distance outside the fence, but situated in the same plane as the band-B dipole elements. Positions 11 through 14 are behind the centers of the antenna arrays about 5 ft outside the exclusion fence. For those points, the approximate distances from the backscreen are: 110 ft for band B, 115 ft for band D, 40 ft for band E, [illegible] ft for band C.

transmitted frequencies were used--one near the center of each bands. They were 7.83 MHz for band B, 10.55 MHz for band C, for band D, and 19.20 MHz for band E. The radar was operated at these fixed frequencies, instead of its usual frequency

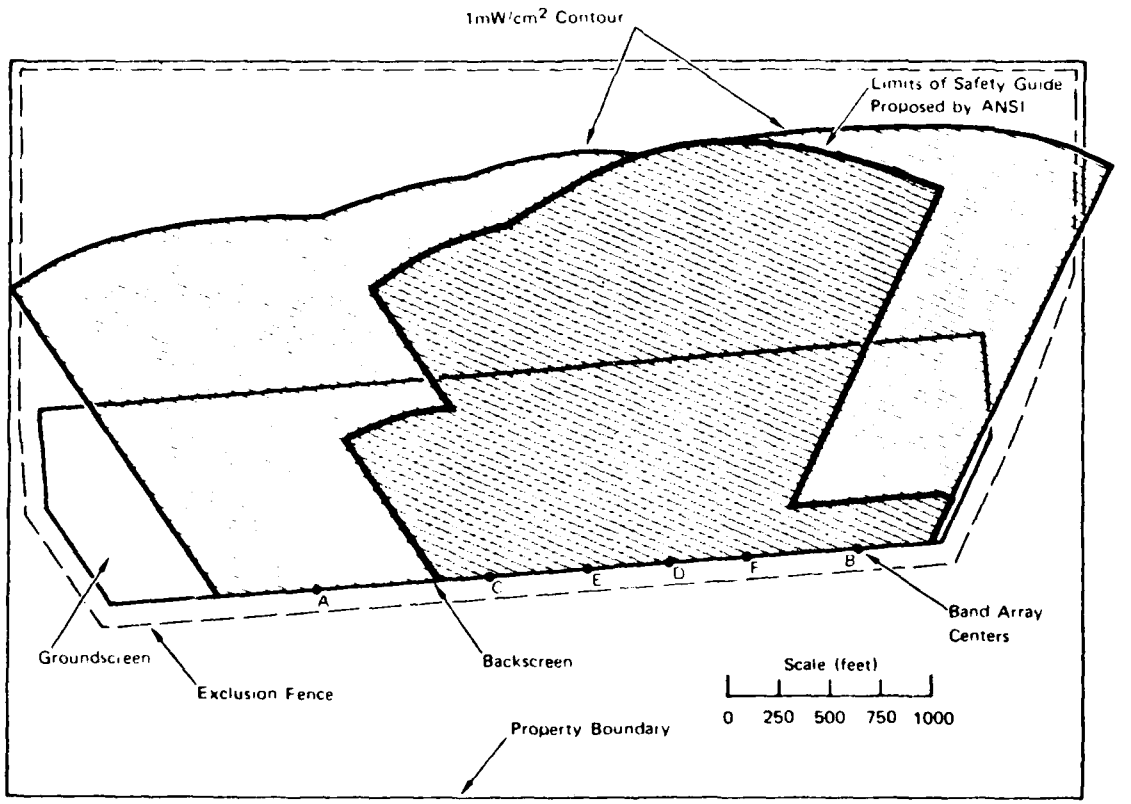


FIGURE A-6 SITE DIAGRAM WITH CONTOURS FOR 1mW/cm^2 AND FOR LIMITS OF SAFETY GUIDE PROPOSED BY ANSI

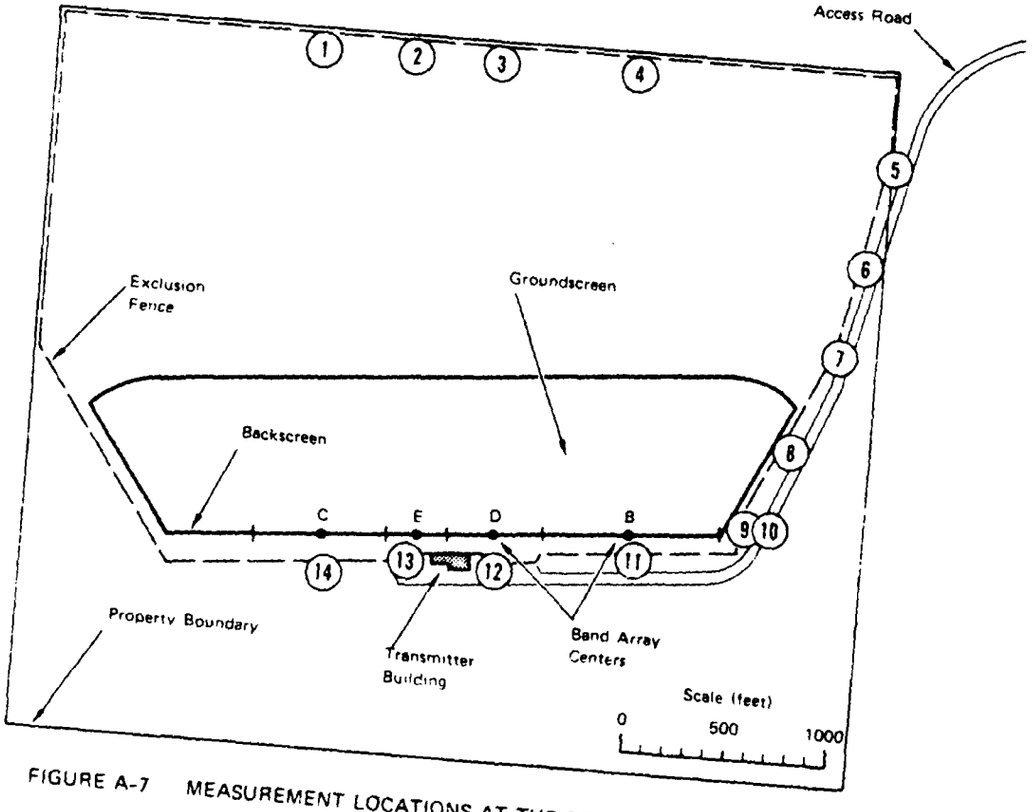


FIGURE A-7 MEASUREMENT LOCATIONS AT THE ERS TRANSMITTER SITE

modulated mode. This procedure facilitated the accurate tuning of the measurement equipment while leaving the power density unaffected.

A.6.2 Measurement Equipment

The measurement receiver was a Singer model NM-26T electromagnetic noise meter operating as a calibrated, tuned radiofrequency voltmeter. This battery-operated receiver has a bandwidth of about 3.6 kHz and is tunable over the range from 150 kHz to 32 MHz. It has an internal impulse generator for calibration at the frequency of measurement. Its rms detector was used so that true rms signal-level values were obtained.

The antenna was a Singer Model 92200-3 loop antenna, which includes its own matching network. The antenna, mounted on a wooden tripod at a height of about 4.5 ft, was connected to the receiver by a 6-ft length of RG-58/U coaxial cable. Although such a loop antenna senses the magnetic field, the manufacturer's calibration curves allow the antenna's output voltage to be interpreted as the equivalent electric field anywhere outside the reactive nearfield (at least one wavelength from the antenna elements). Power density may be calculated from the electric and magnetic field strengths.

Communication between the measurement team and the operators of the radar was provided by VHF handy-talkies.

A.6.3 *Measurement Procedures*

At each of the measurement locations the equipment was set up and communication was established with the radar operators to tell them the desired frequency and beam-slew angle. The receiver was tuned to the appropriate frequency, which was verified by having the transmitter turned off and on. The loop antenna was then oriented for the maximum signal level. It was found that in most cases the maximum signal was found with the plane of the loop vertical and intersecting the antenna array's midpoint; exceptions are discussed later. In this orientation the horizontal magnetic field (vertical electric field) can be measured. Measurements were also made with the plane of the loop horizontal to sample the vertical magnetic field (horizontal electric field). A data sheet was used for each location to log details of the location as well as the meter readings and other pertinent data.

At the far locations (positions 1-4), heavy brush and sloping terrain made it difficult to determine when the measurement team was directly in front of each of the antenna arrays. So, to ensure that the beam maximum was measured, the beam was deviated $\pm$16 degrees in azimuth while measurements were made at 4 degree intervals.

At positions 5 through 8, only band B was measured and the beam was slewed from 30 degrees right azimuth to 4 degrees left while, again, measurements were made at 4-degree intervals.

All four bands were measured at position 9, although the beam was slewed only for the band-B measurements. At position 10, only band B was measured; the beam was slewed 30 degrees right azimuth.

At positions 11 through 14, behind the arrays, the measurements were made with the associated transmitting antenna beams undeviated. The loop was oriented to maximize the level of the received signal.

The ground in the vicinity of the radar was very wet during the time of the measurements, maximizing the soil conductivity and, hence, the power density near the ground. At some of the points there was standing or running water to a depth of perhaps a foot.

The transmitter system was not operated at the full 1.2-MW output; the actual output power was recorded for each of the measurement frequencies so that the measured signal levels could be adjusted upwards to determine the equivalent 1.2-MW output values.

A.6.4 Results

The horizontal magnetic field (vertical electric field) predominated over the vertical magnetic field by a factor of 100 or more at all of the measurement points. This implies that essentially all the power in the field can be determined by a measurement of the horizontal magnetic field, or, equivalently, the corresponding vertical electric field. The meter readings made in the field were converted to power density and then scaled up by the ratio of 1.2 MW to the actual transmitter output power. This scaling factor was determined by the radar crew; it varied with frequency and so a different factor was needed for each of the four measurement frequencies.

Table A-5 shows the measured (and scaled) power density figures and the corresponding calculated values. At positions 1-4, distinct beam maxima were found as the beam was slewed; those maxima are reported on the table. At positions 5-8, the maximum was found when the band-B beam was slewed its full 30 degrees to the right; those values are the ones reported on the table. At position 9, measurements were attempted from all four of the arrays. However, the levels from bands E and C were very low; those arrays presumably direct a deep null in the direction of position 9. Band D also provides a low level there; the level on the table was measured with the beam slewed 30 degrees right. The level from the band-B array fluctated only slightly as the beam was slewed. At position 10 only band B was measured and the beam slew was 30 degrees right azimuth.

Although positions 11 through 14 are each directly behind their respective array centers (between the sixth and seventh elements), the maximum power density was not generally found by directing the loop antenna at the array center. Viewed from behind, the elements of bands D and C are tilted 45 degrees clockwise, those of band B are tilted 45

Table A-5

COMPARISON OF MEASURED AND CALCULATED POWER DENSITIES FOR THE ERS TRANSMITTER

Measurement Position[a]	Band	Frequency MHz	Power Density mW/cm^2 Measured (and Scaled)	Power Density mW/cm^2 Calculated
1	C	10.54	0.035	0.30
2	E	19.20	0.16	0.32
3	D	14.54	0.074	0.22
4	B	7.83	0.088	0.57
5	B	7.83	0.068	0.70[b]
6	B	7.83	0.19	1.1[b]
7	B	7.83	0.14	1.7[b]
8	B	7.83	0.39	3.9[b]
9	B	7.83	0.081	0.30[c]
9	D	14.54	0.0093	0.25[c]
9	C	10.54	7.1×10^{-11}	0.089[c]
9	E	19.20	2.4×10^{-11}	0.29[c]
10	B	7.83	0.12	0.30[c]
11	B	7.83	0.049	0.12[d]
12	D	14.54	0.076	0.18[d]
13	E	19.20	0.38	1.3[d]
14	C	10.54	0.069	0.15[d]

[a] See the diagram of Figure A-7.

[b] The calculated value is a maximum for the beam center, lower values may be encountered near the edge of the beam.

[c] The calculated value results from the specification that sidelobes are at least a factor of 20 smaller than mainlobe maxima.

[d] The calculated value results from the specification that backlobes are at least a factor of 100 smaller than mainlobe maxima.

degrees counterclockwise, and only the elements of the band-E array are vertical. Thus, in the clockwise-tilted arrays, the dipole elements to the right of the measuring point appear foreshortened so that they are directing less than their full strength at the measuring position; however, those elements to the left are viewed in essentially their full length, and so they direct a greater amount of power toward the measurement position. It was confirmed at all four of the behind-the-array points that the maximum power came from the described direction; since the band-E elements are not tilted, the maximum power at position 13 was found with the measurement antenna directed at the array center.

A.6.5 Conclusions

On the basis of the measurements carried out at the ERS site and the corresponding power density calculations, it may be concluded that the methods of calculation of this appendix are adequate to describe and set an upper bound on the power densities of the OTH-B radar system. Specifically, Figures A-3, A-4, and A-5 comprise an adequate description of the largest power densities that may be expected near the ground in the vicinity of the OTH-B transmitter.

A.7 References

Hansen, R. C., Microwave Scanning Antennas, Vol. 1, "Apertures," Academic Press, New York (1964).

Norton, K. A., "The Propagation of Radio Waves over the Surface of the Earth and in the Upper Atmosphere, Part I," Proc. IRE, Vol. 24, pp. 1367-1387 (1936).

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Appendix B

ELECTROMAGNETIC INTERFERENCE HAZARDS

B.1 Introduction

This appendix discusses the potential effects of the OTH-B radar transmitter electromagnetic (EM) fields on equipment other than telecommunications systems. They are termed potential hazards because they describe two potentially dangerous situations that high amplitude radiofrequency (RF) fields can cause under certain circumstances: interference with the normal operation of implanted cardiac pacemakers, and accidental detonation of electroexplosive devices (EEDs). This information significantly amends the EIS, principally in Appendix A, pp. 52, 55, and 56; Section 1.b.(2)(a)7, p. 4; Section 1.e.(3), p. 12; Section 3.d.(2)(a), p. 21; and Section 3.d.(2)(c), pp. 23 and 24.

B.2 Cardiac Pacemakers

B.2.1 Background

Cardiac pacemakers are potentially subject to electromagnetic interference (EMI), and there may be concern that the OTH-B transmitter could affect pacemaker wearers on the ground near the radar. No significant risk to cardiac pacemaker wearers is now expected at electric field strengths less than 200 V/m (equivalent to 10 mW/cm2 power density), in contrast with the 50 V/m susceptibility limit that was used in the 1975 EIS (Department of the Air Force, 1978a). This 4-fold improvement in shielding from electric fields corresponds to a 16-fold increase in power density that current pacemaker units have been measured to withstand (Hardy, 1979). Moreover, pacemakers are less susceptible at the frequencies used by the OTH-B radar than at higher frequencies, and are less susceptible to the frequency modulation used by the OTH-B than to amplitude modulation, particularly the pulse modulation used by most radars (Hardy, 1979).

B.2.2 Safe Separation Limits for Pacemakers Near OTH-B Radar

Because air space in the vicinity of the radar site is restricted, and because airborne exposure would be brief in any event, the main concern is over potential hazards to pacemaker wearers at ground level. Appendix A describes the OTH-B radar transmitter site, its antenna configuration, and its operating parameters. Power density curves at ground level are given for each antenna array in Figures A-3, A-4, and A-5. These data were used to calculate safe separation distances.

Safe separation distances for pacemakers that meet or exceed the specification of 200 V/m (10 mW/cm^2) are depicted in Figure B-1. The power density exceeds the specification only in the mainlobe.

Note that the pacemaker boundaries cover only a portion of the area within the exclusion fence. As noted, this boundary applies to pacemakers that meet the 200 V/m specification. Because all personnel are denied access to the area within the exclusion fence when the radar is operating, pacemaker wearers will not be exposed to this hazard area.

B.3 Electroexplosive Devices (EEDs)

B.3.1 Types of EEDs

EEDs are used to activate secondary explosive charges, to ignite propellant systems, and to actuate electroexplosive switches. Perhaps the most common EED is the ordinary electrical blasting cap. The four basic types of EEDs are described below (Hovan, 1978):

- Exploding bridgewire: Requires a high energy capacitive discharge pulse to explode bridgewire.
- Normal bridgewire: An explosive mix is glued to the bridgewire, which is heated by electrical current to detonate the adhesive primer.
- Composition mix: Electrical current is passed through a conductive explosive mix to ignite it.
- Carbon bridge type: This is used internally in three or four weapons systems and in 20-mm cartridge primers; it is the EED type most sensitive to RF fields, and it is also sensitive to static electricity; the hazard for the 20-mm primer comes from ground crews touching the base primer during loading; if RF energy (or static electricity) is present, personnel touching the primer can couple energy into the EED.

B.3.2 Safe Separation Distance Criteria

EEDs are susceptible to ignition by exposure to EM fields. The degree of susceptibility depends on many variables: the safe no-fire threshold of the EED, the ability of the EED leads to capture RF energy, the frequency and average power density of the RF energy, and the condition of exposure of the EED--whether it is contained in a shielded cannister, mounted inside an aircraft whose skin provides partial shielding, or exposed to the environment without shielding. The safe exposure criterion is expressed as a safe power density, in W/m^2, or as a safe separation distance. It should be noted that as the distance, d, between an EED on the ground and the RF transmitter is increased, the power density at the EED decreases as $1/d^n$, where n varies from 0.7 to 4, depending on band and range (see Appendix A).

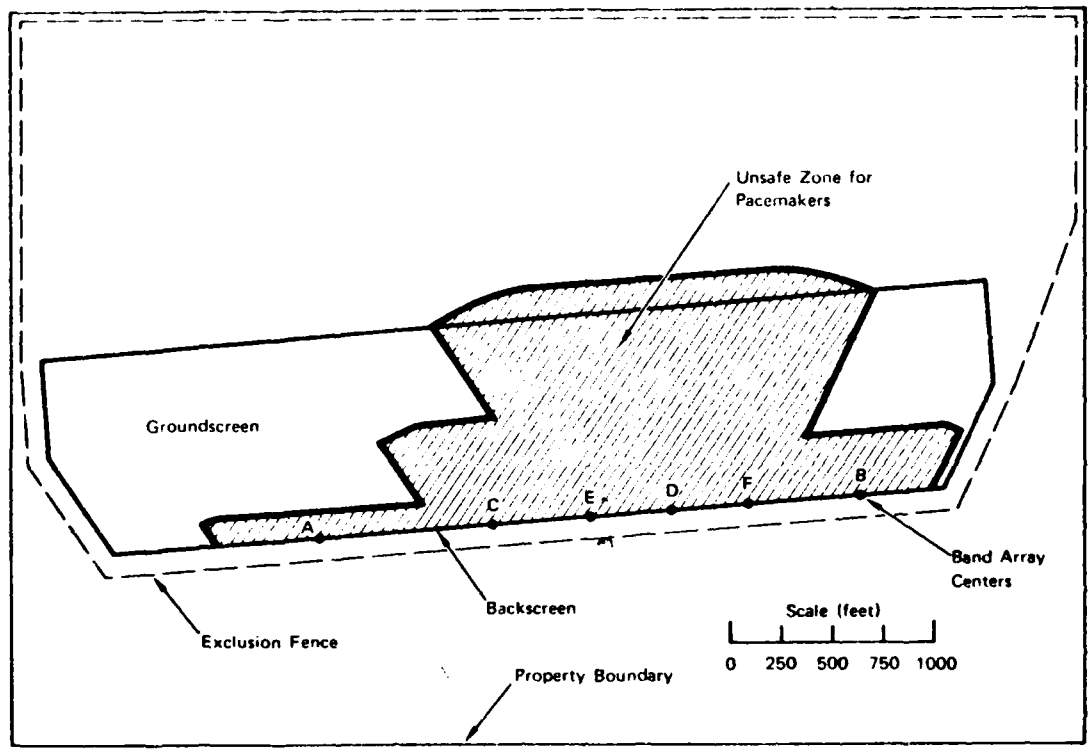

FIGURE B-1 SAFE SEPARATION DISTANCES FOR PACEMAKERS

The safe separation distances specified by Air Force Regulation AF 127-100, "Explosive Safety Standards" (Department of the Air Force, 1978b), applies to storage, transport, and loading of EEDs, and to aircraft with EEDs while they are taxiing and in flight. The distances are based on a worst case situation, that is, on the most sensitive EED currently in inventory, shielded or unshielded, with its leads or circuitry inadvertently formed into a resonant antenna. Again, note that the standard is based on a conservative, no-fire threshold for the EED. Exposure exceeding that threshold does not imply that the EED will fire. The actual firing threshold of the EED is several orders of magnitude greater than the safe no-fire threshold.

B.3.3 EM Field Safety Standards for EEDs

The AF 127-100 (Department of the Air Force, 1978b) criteria for safe power density exposure for EEDs are summarized in Table B-1. All safe exposure limits are given in terms of average power density. Air Force safety standards for EEDs apply to the manufacture, testing, storage, transport, loading, and operation of systems containing EEDs. The standards have been established to minimize the accidental detonation of weapons by environmental causes or by personnel. In addition, AF 127-100 considers various electrical hazards, including location of power lines and electrical equipment, lighting, static electricity and grounding, and lightning protection.

B.3.4 EEDs Near OTH-B Transmitter Site

No EEDs will be handled, stored, or assembled at the OTH-B radar transmitter site. No 20-mm ammunition is stored at the site and no airport is on or near it. The presence of EEDs on overhead aircraft cannot be ruled out, but existing regulations restrict all aircraft from entering the potentially hazardous areas.

B.3.5 EED Susceptibility to OTH-B Fields

From the considerations in paragraphs B.3.3 and B.3.4, three individual safe separation boundaries must be determined for EEDs, in accordance with the following standards:

(1) Exposed EEDs, leads form antenna: 0.001 mW/cm^2

(2) EEDs in storage or transport in nonmetallic containers, leads shorted: 0.026 mW/cm^2

(3) EEDs on aircraft in flight: 10 mW/cm^2

Table B-1

SAFE EXPOSURE LIMITS FOR EEDs AT OTH-B FREQUENCIES

Exposure or Storage Condition for EED	Average Power Density W/m^2	Average Power Density mW/cm^2
EEDS in exposed condition, leads formed into antenna	0.01	0.001
Leadless EEDs (20-mm ammunition handling criteria)	0.01	0.001
EEDs in storage or transport in nonmetallic containers, leads shorted	0.26	0.026
Aircraft taxiing with externally loaded weapons	6.63	0.663
EEDs stored and transported in metallic containers	100	10
Inflight aircraft with externally loaded weapons	100	10
Shipment of EEDs inside cargo aircraft	100	10

Source: Department of the Air Force (1978b).

Safe separation criteria for EEDs were calculated from the information in Appendix A and the EED standards, as they were in Section B.2.3 for pacemakers. For the last category, EEDs on aircraft in flight, the safe separation distance is a slant range of 1,250 ft in front of the radar (bands E and F). Given the elevation limits of the mainlobe, an aircraft altitude of 550 ft above the radar (2,000 ft above mean sea level) would be safely separated. The hazard volume is well within the volume from which all aircraft are restricted.

The situation for the first two EED categories is shown in Figure B-2. The OTH-B power densities at ground level exceed the safety standard for distances well beyond the exclusion fence for most azimuths. Tables B-2 and B-3 show the safe separation distances for the [illegible] of all antennas. The tables indicate that the restricted area for exposed EEDs (leads form antenna) extends nearly 3 miles toward the

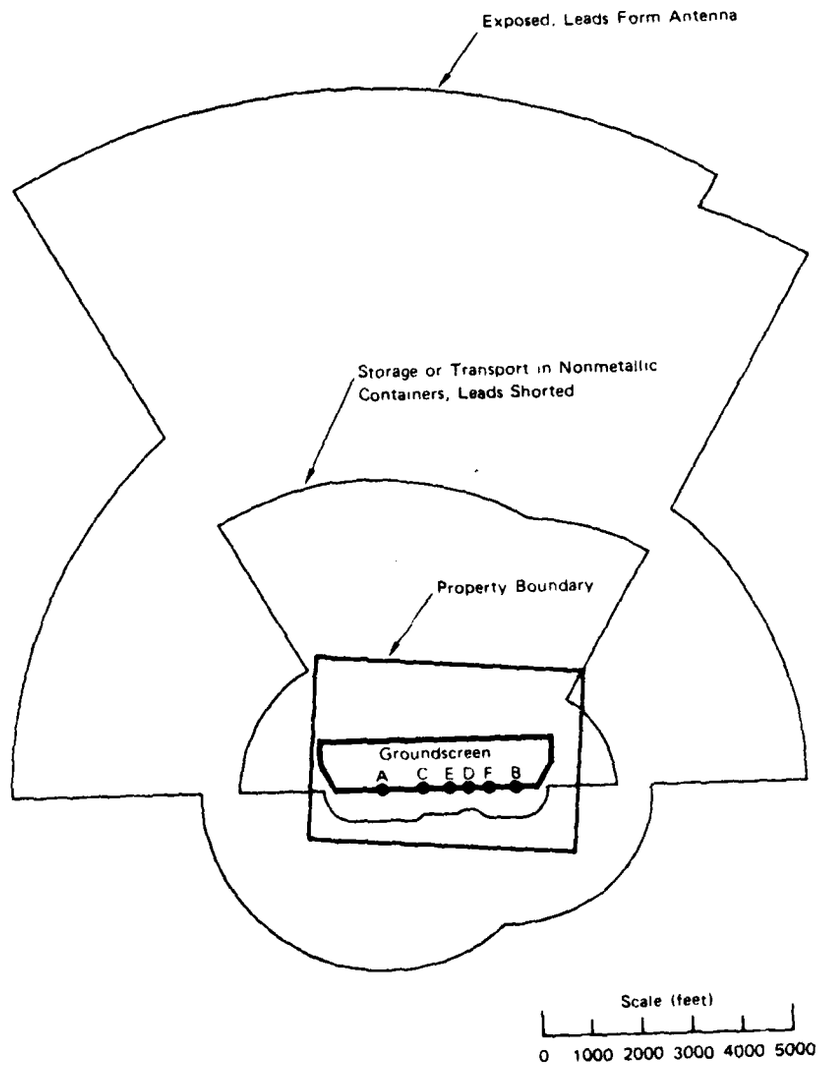

FIGURE B-2 SAFE SEPARATION DISTANCES FOR EEDs

Table B-2

SAFE SEPARATION DISTANCES FOR EEDs IN STORAGE OR TRANSPORT IN NONMETALLIC CONTAINERS, LEADS SHORTED (feet)

Band	Mainlobe	Left Sidelobe	Right Sidelobe	Backlobe
A	6,200	2,800	3,500	610
B	5,400	3,800	2,000	580
C	4,800	2,700	3,000	510
D	4,500	3,300	2,300	430
E	5,100	3,600	3,100	510
F	5,100	4,200	2,300	440

Table B-3

SAFE SEPARATION DISTANCES FOR EXPOSED EEDs, LEADS FORM ANTENNA (feet)

Band	Mainlobe	Left Sidelobe	Right Sidelobe	Backlobe
A	14,000	6,600	8,000	3,600
B	12,100	8,700	4,800	2,700
C	10,900	6,200	6,800	2,100
D	10,100	7,500	5,200	1,600
E	11,400	8,200	7,100	1,500
F	11,400	9,500	5,300	1,200

front of the radar, substantially more than a mile to the sides, and about one-half mile behind the antennas. The safe boundaries for EEDs in storage or transport in nonmetallic containers (leads shorted) are less than half as far from the radar as the boundaries for exposed EEDs (leads form antenna). Figure B-3 shows the composite safe separation boundaries for EEDS for the 3-sector 180-degree system as currently planned.

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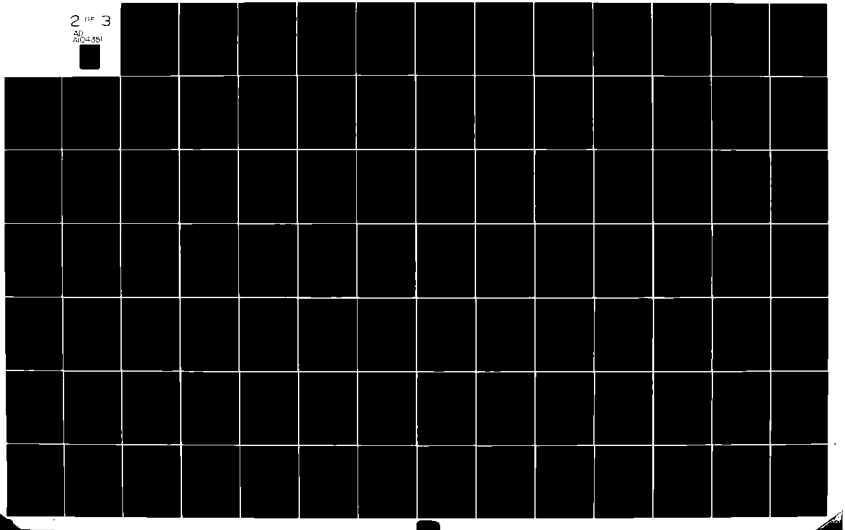

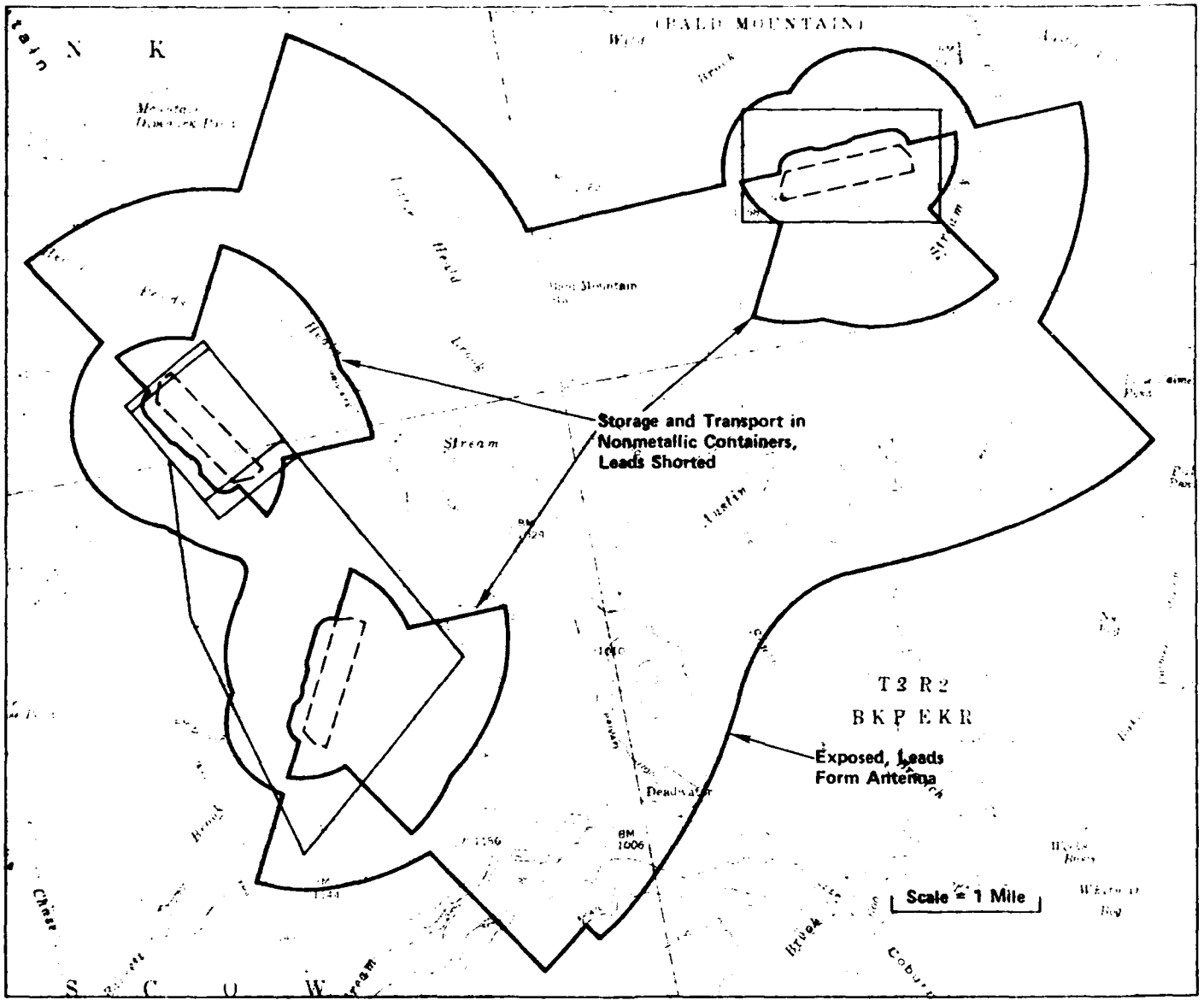

FIGURE B-3 SAFE SEPARATION DISTANCE CONTOURS FOR EEDs WITH PLANNED TRANSMIT SITE CONFIGURATION

B.4 References

Department of the Air Force, "Exposure to Radiofrequency Radiation," AFOSH Standard 161-9 (10 October 1978a).

Department of the Air Force, "Explosive Safety Standards," AF Regulation 127-100 (March 31, 1978b).

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Appendix C

HUMAN EXPOSURE TO RADIOFREQUENCY RADIATION

C.1 Introduction

This appendix supersedes all human health information in the EIS, particularly in paragraphs 1.e(3) and (4), pp. 11-12, 2.d(2), pp. 21-22, and in Appendices A, B, and C.

C.1.1 Background

C.1.1.1 Definition of "RFR"

The generic term radiofrequency radiation (RFR) is used herein to include other terms commonly found in the literature, such as electromagnetic radiation (EMR), nonionizing electromagnetic radiation (NIEMR), microwave radiation, radiofrequency electromagnetic (RFEM) fields, electromagnetic fields (EMF), microwave fields, and others. The term RFR, as used here, is intended to apply to frequencies up to about 18,000 MHz (18 GHz). The center frequencies of the OTH-B radar range from 5 to 30.6 MHz, mostly within the 3-to-30 MHz High-Frequency (HF) band.

C.1.1.2 Purpose of this Appendix

The primary issue addressed in this appendix is whether brief or continual exposure of people to the power densities of RFR produced by OTH-B is likely to affect their health adversely. This issue has been examined in depth by conducting a critical review of the literature on biological effects of RFR. Documents were selected from the large body of literature on the subject and analyzed. The selection included those that are most significant scientifically and most pertinent to the power densities of RFR likely to be encountered in the geographic region around the radar.

C.1.1.3 Exposure Levels

Calculations of the maximum power densities in the mainlobe at the exclusion fence yielded approximately 0.6 mW/cm^2 for band A, 0.7 mW/cm^2 for band B, 0.4 mW/cm^2 for band C, 0.3 mW/cm^2 for band D, and 0.5 mW/cm^2 for bands E and F (see Appendix A for band descriptions). Along the right section of the exclusion fence (see Figure A-1, p. A-3, and Table A-4, p. A-12), the power densities for band B at locations 2 and 3 are about 1.1 and 0.3 mW/cm^2, respectively; the values for the other bands at these locations are much smaller because the centers of the antenna arrays for these bands are more distant than that of the band B antenna. Similarly, along the left section of the exclusion fence, the power densities for band A at locations 5 and 6 are about 0.1 and 0.8 mW/cm^2, respectively. Along

the rear section of the exclusion fence, the highest power density is for band F at location 4; the value there is about 0.6 mW/cm^2.

At Chase Pond, about 1.25 miles southwest of the ERS site and site of the first full 60-degree sector, where several private camps exist, the calculated power density is less than 0.0001 mW/cm^2 for the full 180-degree system. At the closest population centers of Moscow and Bingham, nearly 6 miles south of the closest 60-degree sector, the calculated power density is less than 0.00001 mW/cm^2 for the planned 180-degree system, and less than 0.0001 mW/cm^2 if plans change such that mainlobes rather than sidelobes are directed to the area. Similarly, at Caratunk, over 7 miles northwest of the closest sector, the calculated power density is less than 0.0000001 mW/cm^2.

C.1.2 Data Base and Literature Selection

Many sources were used in acquiring a working data base for this assessment, including bibliographies provided in previous reviews of the literature; a comprehensive bibliography prepared by U.S. Government personnel; published proceedings of recent seminars and meetings on the biological effects of RFR; the computerized data base on Biological Effects of Electromagnetic Radiation (BEER file) of the Mead Technology Corporation, Dayton, Ohio; and compilations of articles published by the Franklin Institute. Abstracts of papers presented at recent symposia on the biological effects of RFR were also reviewed.

Several criteria were used in selecting articles for inclusion in this review. Preference was given to complete papers published in scientific journals or proceedings of scientific symposia. Abstracts of presentations at recent scientific symposia were also selected if they included adequate details of the procedures and findings. Criteria included the date of publication (more recent articles were preferred because of improvements in experimental methodology and in the technology of exposure and dose measurement) and the significance of the findings to human health (e.g., studies of human populations to ascertain whether the occurrence of specific effects is statistically higher in population samples exposed to RFR than in similar population samples not exposed, and experiments involving long-term exposure of animals). Other criteria included the relevance of an article to others on the same topic and possible relevance to concerns expressed by citizens' groups. We stress that most bioeffects of RFR are frequency dependent but not frequency specific per se; rather, the frequencies of the incident RFR (together with its average power density and polarization and the size, shape, and orientation of the biological entity) determine the rate of energy absorption and its internal distribution. For this reason, the selection of articles was not confined to those involving frequencies close to the OTH-B radar frequencies, but was extended to articles involving frequencies over the general range from 0 to 18 GHz as appropriate. The number of articles selected was necessarily limited because of resource constraints. However, we consider the articles selected to be representative of the large number of documents related to the biological effects of RFR.

C.1.3 Eastern European Bioeffects Literature

Probably the most controversial aspects of research on the biological effects of RFR are the large discrepancies between results, at low levels of RFR, reported in the Eastern European literature and those obtained in Western countries such as the United States, and the basic differences in philosophy between the two groups of countries in prescribing safety standards or guidelines for the protection of humans against possible hazards from exposure to RFR.

From the end of World War II to about the late 1960s, few of the scientific reports on bioeffects research in the USSR (or other Eastern European countries) were amenable to critical review because they lacked essential information. In the early 1970s, starting essentially with an international conference on the bioeffects of RFR in Warsaw in 1973 under the joint sponsorship of the World Health Organization (WHO), the U.S. Department of Health, Education, and Welfare (HEW), and the Scientific Council to the Minister of Health and Social Welfare of Poland, international interchanges of information increased materially, and translations of Eastern European articles became easier to obtain. Because most of the Eastern European documents published before 1973 (and many since then) are merely abstracts that contain no details of the experimental method, number of subjects, or analytical approach used in the study, evaluation of them proved difficult. More recent Eastern European studies contain more detail, and some of them have been included in our analysis.

C.2 Present Climate and Context

C.2.1 Proliferation of RFR Emitters

Public use of RFR-generating devices and acceptance of their benefits have been growing almost exponentially over a number of years. Public television and radio broadcasting stations, ham radio transmitters, citizens band radios, ground level and satellite communication systems, civil and military aircraft navigation systems, airport traffic control systems, medical diathermy units, defense tracking systems, remote garage door opening devices, microwave ovens, and a variety of units for industrial heating and processing of materials contribute to the expansion of RFR use in this country.

Growth in demand for the services provided by these devices is reflected by the following figures for the United States (McRee, 1978). During 1976, sales of communications and electronics products are estimated to have totalled $38 billion. By the end of 1977, the Federal Communications Commission (FCC) had authorized transmissions by more than 9 million transmitters. Between 1971 and 1978, the number of FM stations operating in the 88-108 MHz band increased by 87%. The National Institute of Occupational Safety and Health (NIOSH) has estimated that more than 35 million industrial RFR sources for heating and drying are in use in the plastics, paper, and other industries. Approximately 5 million microwave ovens have been installed in homes in

the United States (McConnell, 1978). By the end of 1979, about 30 million citizen band (CB) radios had been licensed, with those operating in the 27-MHz band, each capable of emitting up to 4 watts. Domestic and business satellite communications systems are burgeoning. Air and maritime navigation systems make widespread use of fixed and mobile radars.

All of these devices are regulated by the federal government, mainly the FCC, and all are restricted to specific frequency bands. The power levels that most devices may emit are also restricted. Still, as the number of such devices increases, the background level of RFR in this country, particularly in urban and industrial centers, is bound to increase as well. It is therefore appropriate to ask whether this increasing level of RFR will be deleterious to human health.

Various agencies of the federal government have established programs to deal with the question of effects of RFR on human health. The Environmental Protection Agency (EPA) is conducting a study of environmental levels of RFR. The Bureau of Radiological Health (BRH) has promulgated a performance standard for permissible microwave oven leakage (21 CFR 1010, "Performance Standards for Electronic Products"). NIOSH is investigating the use of industrial microwave devices. All three of these agencies, together with the Department of Defense (DOD), maintain research programs on the biological effects of RFR, with the objective of assessing effects on human health. The results of these programs indicate that the biological effects of RFR are largely confined to average power densities of thousands and tens of thousands of microwatts per square centimeter. Further, present maximum environmental levels in cities are generally in the range of 0.01 to 5 microwatts/cm^2, with the occasional exception of regions in the vicinity of broadcast towers, where environmental levels may range from 10 to higher than 200 microwatts/cm^2 (see Section C.2.2). Thus, there is no credible scientific evidence that present environmental levels of RFR are likely to adversely affect the health of the general population.

In summary, there is a widespread climate of acceptance of the benefits of RFR devices for communications, radar, personal and home use, and industrial processes. On the other hand, there is concern that the proliferation of the use of RFR devices, including OTH-B, may be associated with some as-yet-undefined hazardous biological effects. The purpose of the present document is to address such concerns as they pertain to OTH-B.

C.2.2 Measurements of Environmental Levels of RFR in Selected U.S. Cities

EPA is measuring the environmental field intensities at selected locations in various U.S. cities. Tell and Mantiply (1980) and Janes (1979) discuss the results for the 15 cities (a total of 486 sites) studied so far. The sites in each city were selected to permit estimations of cumulative fractions of the total population being exposed at or below various average power densities, based on the population figures for the 1970 census enumeration districts.

Field intensity measurements were made at 6.4 m (20 ft) above ground at each site in the following frequency ranges (Janes et al., 1977): 0.5 to 1.6 MHz (the standard AM-radio broadcast band), 54 to 88 MHz and 174 to 216 MHz (the VHF-TV bands), 88 to 108 MHz (the standard FM-radio broadcast band), about 150 and 450 MHz (land-mobile bands), and 470 to 890 MHz (the UHF-TV bands). The signals in each band were received with separate antennas designed specifically for each band. However, the measurements in the standard AM-radio broadcast band were not included in the analyses because this band is below the 10 MHz lower frequency limit of the present U.S. radiation protection guideline (ANSI, 1974).

The measured field strengths at each site were integrated over the frequency bands from 54 to 890 MHz included in the analyses and converted into equivalent average power densities. The site values in each city were then used with the population figures in the various census enumeration districts in a statistical model designed to estimate the population-weighted median exposure value for that city and to calculate other statistics of interest.

The population-weighted median value for a city is defined as the average power density at or below which half the population of the city is being exposed. The estimates are based on the assumption of continuous exposure of people at their place of residence; they do not take into account population changes since the 1970 census, population mobility, exposure at heights greater than 6.4 m, attenuation of signals by buildings, or periods of time when any of the contributing RFR sources are not transmitting. These median values range from 0.002 microwatts/cm^2 (for Chicago and San Francisco) to 0.020 microwatts/cm^2 (for Portland, Oregon). The population-weighted median for all 15 cities is 0.0048 microwatts/cm^2. Also, the percentage of the population of each city exposed to less than 1 microwatt/cm^2 ranges from 97.2% (for Washington, D.C.) to 99.99% (for Houston, Texas), with a mean value for all 15 cities of 99.4%. The major contributions to these exposure values are from the FM-radio and TV broadcast stations.

EPA also measured RFR levels at sites close to single or multiple RFR emitters, e.g., at the bases of transmitter towers and at the upper stories (including the roof) of tall buildings or hospital complexes close to transmitter towers. At the base of an FM tower on Mt. Wilson, for example, the fields ranged from 1,000 to 7,000 microwatts/cm^2 (Tell and O'Brien, 1977, cited in Janes, 1979), but such values are believed to be uncommon. Most measurements in tall buildings close to FM and TV transmitters yielded values well below 100 microwatts/cm^2, but a few values were close to or slightly exceeded 200 microwatts/cm^2 (e.g., 230 microwatts/cm^2 on the roof of the Sears Building, Chicago).

Janes (1979) also discussed the field intensities near ground-based transmitters of satellite communications systems, radars used for air-route surveillance and other activities, microwave radio relay transmitters, microwave ovens, and personal radios (CBs). He also mentioned other emitters such as those used for medical (diathermy,

electrosurgery) and industrial (heating, drying, and sealing) applications.

Because OTH-B transmitting sites are likely to be distant from major population centers such as those analyzed in these studies, the increases in population exposure values in the areas studied by EPA and other densely populated regions due to operation of OTH-B are likely to be very small.

C.2.3 Problems of Risk Assessment

The assessment of risk to human health and the setting of standards to protect health are extremely complex problems. In addition to purely technical and scientific questions, there are problems of philosophy, law, administration, and feasibility of programs that are still only vaguely recognized. It is clearly beyond the scope of this document to deal with those subjects in detail, but it is important that they be mentioned. This appendix will concentrate on three aspects of the present issue: the scope of biological effects considered in setting standards, the overall approach to setting standards, and standards of protection from overexposure to RFR in the United States, the USSR, and other countries.

Alternative approaches to determining the acceptable degree of risk or undesirable effect can be illustrated by comparing occupational air pollution standards that prevailed until recently in the USSR and the United States (Zielhuis, 1974). In the USSR, maximum allowable concentrations (MACs) for airborne noxious agents are set at a value that will not produce any deviation from normal in physiological parameters, or any disease in anyone exposed to the agent (occupational or general population). In the United States, threshold limit values (TLVs) for airborne noxious agents are set to ensure that nearly all workers can be exposed regularly during the working day without adverse effect. The differences stand out clearly: in the USSR, all biological effects are considered without regard to their medical significance or the possibility of human adaptation, and the values of MAC selected must, in principle, protect the most susceptible member of the population. In the United States, only harmful effects are considered, and protection is not extended to the most susceptible workers, except that a safety factor is generally included in the TLV such that an adverse reaction in an individual can be detected before serious medical consequences ensue.

Both of the approaches in the preceding paragraph are predicated on the existence of a threshold concentration; that is, on a concentration below which no biological effect will occur. In the absence of a true threshold, one can only weigh the extent of protection to give to the population against the cost and technical feasibility of providing that protection. Making such choices is the function of risk/benefit analysis in the assessment of environmental hazard.

The subject of the existence or nonexistence of thresholds has been debated at length, but much of the debate has been conducted on the basis of opinion rather than evidence. As a practical scientific matter, thresholds for noxious or deleterious effects must exist for at least some substances, because many naturally occurring substances are essential to life at one concentration and highly toxic at higher concentrations (Horne, 1972). In this document, the possible existence of threshold levels for RFR effects is considered on a case-by-case basis, with due regard for the physiological mechanisms of effect.

C.2.4 Exposure Standards

This discussion of the exposure standards in various contries is included as general background information. However, it is emphasized that existing or contemplated exposure standards are not at issue, and do not form the basis for any of the recommendations contained in this document. Instead, the basis for decision is the presence or absence of documentable evidence of potentially harmful effects from exposure to the RFR from OTH-B on the population working or living in the vicinity of the facility.

The term "exposure standards" is generally applied to specifications or guidelines for permissible occupational and/or nonoccupational exposure of humans to electromagnetic fields. The standards are expressed as maximum power densities or field intensities in specific frequency ranges and for indicated exposure durations.

The present U.S. standard is based on average power densities and is essentially the same as the American National Standards Institute Radiation Protection Guide ANSI C95.1 (ANSI, 1974). Under this standard, which applies to the frequency range from 10 MHz to 100 GHz, none of the following values, when averaged over any exposure period of 0.1 hour, should be exceeded:

- Power density: 10 mW/cm^2
- Square of electric field strength (E^2_{rms}): 40,000 V^2/m^2
- Square of magnetic field strength (H^2_{rms}): 0.25 A^2/m^2
- Energy density: 1 $mW\text{-}hr/cm^2$.

The values of E^2_{rms} and H^2_{rms} above are approximately the "free-space equivalents" of 10 mW/cm^2 (100 W/m^2) power density--i.e.,

$$E^2_{rms} = (Z) \times (100\ W/m^2) \qquad (1)$$

$$H^2_{rms} = (1/Z) \times (100\ W/m^2) \quad , \qquad (2)$$

where Z represents the "impedance" or value of E/H for free space, rounded off from 377 ohms to 400 ohms to yield E_{rms} = 200 V/m and H_{rms} = 0.5 A/m instead of values to more than one significant figure. Where only one type of field is present, the appropriate value of maximum field applies. Where both types of field are present (e.g., from separate sources of magnetic and electric fields), the maximum energy-density value above imposes the additional condition that the total contributions from both fields, averaged over any 0.1-hour period, should not exceed the equivalent of 10 mW/cm^2. The rationale accompanying the standard states that these maximum permissible levels are appropriate for exposure under moderate environmental conditions (temperature and humidity); lower levels should be used under environmental conditions that induce significant heat stress.

The 10 mW/cm^2 value originated from the physiological consideration that whole-body exposure of a human to levels of about 100 mW/cm^2 or more would produce a mild to severe (depending on the level) increase in thermal load. A safety factor of 10 was then applied to the lower limit of this power-density range.

The U.S. standard does not contain specific enforcement or punitive provisions for violations. It has been promulgated by the Occupational Safety and Health Administration (OSHA) as a radiation protection guide for occupational exposure and has been adopted by a number of organizations, including the DoD. The principle underlying this guide was the belief, based on the then-available scientific evidence, that nearly all workers could be exposed to RFR at such levels during the normal series of working days without adverse effects. Adoption of the guide gave recognition that electromagnetic fields at the maximum possible levels might cause biological effects that have no medical consequences, or that workers could readily accommodate to such effects.

Based on recent experimental and theoretical results, EPA, NIOSH, and ANSI are considering possible revisions in the U.S. standard. The ANSI Subcommittee C95.4 has proposed the frequency-dependent standard shown in Figure C-1 for both occupational and nonoccupational exposure. It is based on a mean-whole-body specific-absorption-rate (SAR) limit of 0.4 W/kg (see Section C.5.1.2 for the definition of SAR), which includes a safety factor of 10 and is also averaged over any 0.1-hour period. In the range 3-30 MHz, the applicable formula is $900/f^2$, which yields a corresponding range for the maximum allowable average power density of 100 to 1 mW/cm^2. Note that the calculated power densities for the six frequency bands of OTH-B at the exclusion fence are within the values prescribed by this proposed standard; the power densities at nearby population centers are far lower. However, the question of

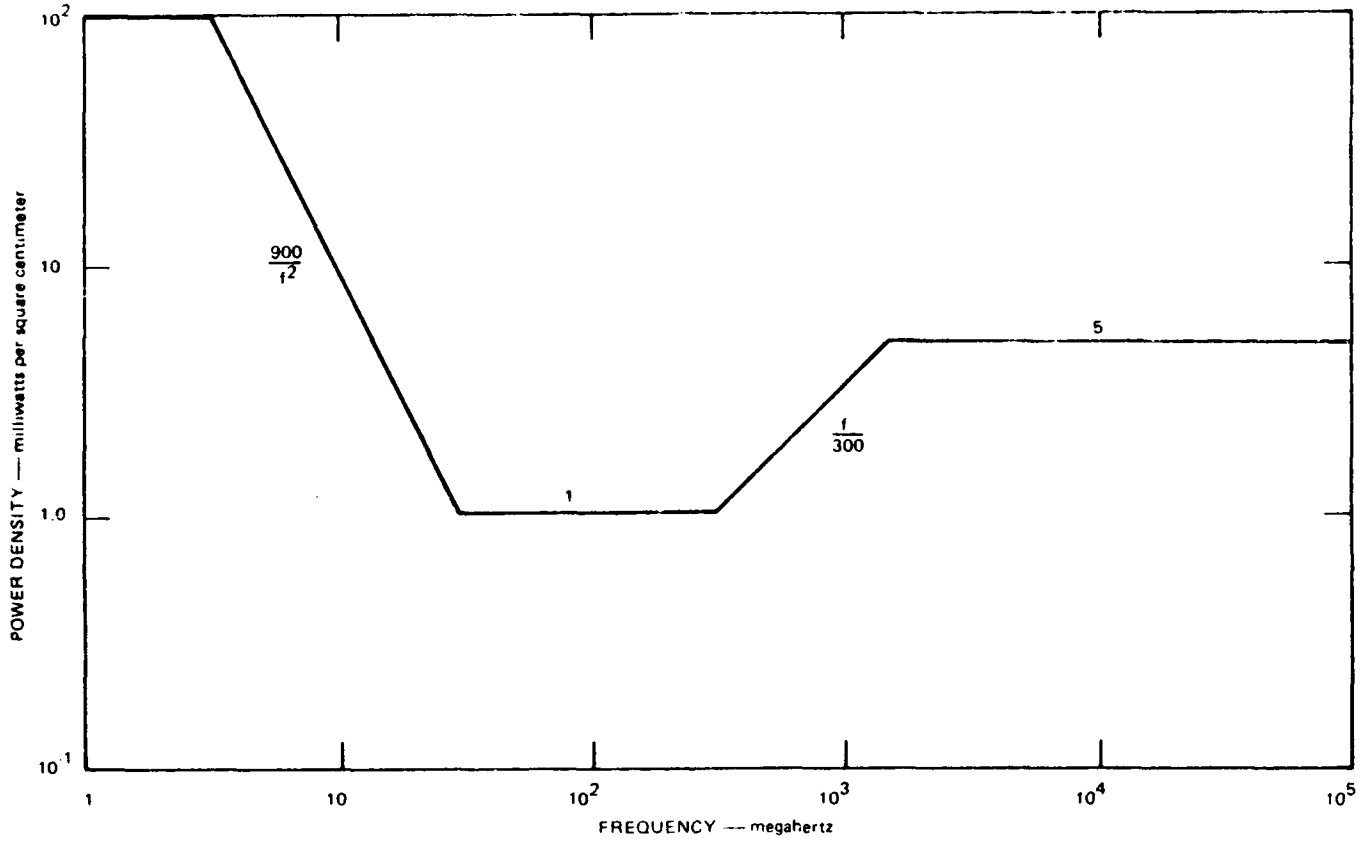

FIGURE C-1 SAFETY GUIDE FOR WHOLE-BODY EXPOSURE OF HUMANS, PROPOSED BY ANSI SUBCOMMITTEE C95.4*

*Based on average SAR limit of 0.40 W/kg in exposed tissue.

environmental standards for the general (nonoccupational) population is still under consideration by EPA and NIOSH.

Present standards in the United Kingdom, France, and West Germany are essentially the same as the current ANSI guideline (Stuchly and Repacholi, 1978). This was formerly true for Canada as well. However, the Canadian federal government has recently revised its standard along the lines shown in Table C-1. The maximum permissible general (nonoccupational) level for continuous exposure is 1 mW/cm^2, applicable to frequencies in the band from 10 MHz to 300 GHz. For occupational exposure, the maximum levels are frequency- and duration-dependent. For example, for the frequency range from 1 to 300 GHz, the new standard permits exposure to 5 mW/cm^2 for a maximum of 8 hours/day, up to 10 mW/cm^2 for 6 minutes or less, and up to 25 mW/cm^2 for 2.4 minutes or less.

Table C-1

NEW CANADIAN
MAXIMUM PERMISSIBLE EXPOSURE LEVELS

Exposure Group	Frequency (GHz)	Duration	Maximum Level
General public	0.01 to 300	24 hr	1 mW/cm^2
Occupational	0.01 to 1	8 hr	60 V/m 0.16 A/m 1 mW/cm^2
		t(min) = 60/P P = power density	1 to 25 mW/cm^2
	1 to 300	8 hr	5 mW/cm^2
		t(min) = 300/P	1 to 10 mW/cm^2
		t(min) = 60/P	10 to 25 mW/cm^2

The Swedish standard, which used to be essentially the same as ANSI C95.1, was revised in 1976 as shown in Table C-2 (Stuchly and Repacholi, 1978). Again, the new maximum occupational exposure levels are about tenfold lower than they were. The new standard is assumed to apply to the general (nonoccupational) population as well as to RFR workers.

Presumably, the reductions of maximum permissible levels in the Canadian and Swedish standards were engendered in part by consideration of some of the relatively recent research results indicative of bioeffects due to chronic exposure at power densities in the range from 1 to 10 mW/cm^2.

In the USSR, the maximum level for 24-hour exposure of the general population is 5 $microwatts/cm^2$ (Shandala, 1978; McRee, 1979). The occupational standard is summarized in Table C-3 (Stuchly and Repacholi, 1978; McRee, 1979). It specifies higher maximum levels than those for the general population. For example, in the frequency range from 10 to 30 MHz, it permits a level of 20 V/m ("free-space-equivalent" of about 100 $microwatts/cm^2$) for a full working day, and presumably does not specify a limit for frequencies below 10 MHz. The Soviet military services and establishments are specifically exempted from such standards. The process by which the USSR standard was arrived at is unknown, because the Council of Ministers that sets standards does not publish its proceedings. Nevertheless, we can surmise that the standard is based in part on the claimed existence of "nonthermal" effects and on the philosophy that exposure to power-density levels that cause any effect is potentially harmful, leading to the application of large safety factors in formulating maximum permissible levels.

C.3 Assessment of Scientific Information

In an assessment of the potential biological effects of RFR from OTH-B, it is necessary to consider certain quantitative relationships among (1) the physical parameters of the RFR such as frequency, power density, and polarization; (2) the mechanisms of absorption and distribution of energy within the biological organism; and (3) the resulting biological effects as measured by some functional or anatomic alteration. Like all scientific theory, the body of biophysical theory that links these three factors has been synthesized from a variety of experimental evidence. The theory is subject to refinement or revision as valid new evidence accumulates that is inconsistent with the theory. Nevertheless, it furnishes the context in which new experimental evidence is considered.

Experimental evidence comes from the observation of experimental animals and, sometimes, humans who have been exposed to RFR. The physical characteristics of the radiation, the mechanisms of interaction, and the biological response are known in some cases, at least qualitatively. Obviously, the most directly applicable

Table C-2

SWEDISH OCCUPATIONAL STANDARD OF
MAXIMUM PERMISSIBLE EXPOSURE LEVELS

Frequency (GHz)	Exposure Duration (hrs)	Maximum Power Density (mW/cm^2)	Remarks
0.01 to 0.3	8	5	Averaged over 6 min
0.3 to 300	8	1	Averaged over 6 min
0.01 to 300		25	Averaged over 1 s

Table C-3

USSR MAXIMUM PERMISSIBLE LEVELS FOR OCCUPATIONAL EXPOSURE

Frequency (GHz)	Exposure Duration	Exposure Limit	Remarks
0.01 to 0.03	Working day	20 V/m	--
0.03 to 0.05	Working day	10 V/m 0.3 A/m	--
0.05 to 0.3	Working day	5 V/m	--
0.3 to 300	Working day	10 microwatts/cm^2	Stationary antennas
	Working day	100 microwatts/cm^2	Rotating antennas
	2 hr	100 microwatts/cm^2	Stationary antennas
	2 hr	1 mW/cm^2	Rotating antennas
	20 min	1 mW/cm^2	Stationary antennas

experimental evidence concerning possible bioeffects of OTH-B would come from experiments in which humans were exposed to its specific frequency range and likely power density values. Furthermore, the best evidence would come from quantitative evaluation of a large number of biological endpoints. Such data, however, do not exist. The available information is indirect because it is derived primarily from experiments with animals and requires at least some extrapolation o. species, field characteristics, duration of exposure, and biological effects.

In retrospective epidemiologic studies, the data are acquired after the event to be studied has occurred. Although such studies deal with human subjects and thus might furnish direct evidence from a species standpoint, epidemiologic evidence is considered to be indirect, for two reasons. First, not only are the estimated exposure parameters usually unlike those of the OTH-B, but numerical values of the exposure parameters for most epidemiologic studies are not known in detail. Second, the extent to which the unexposed control group of people selected for comparison differs from the exposed population (other than in exposure to RFR) is a critical matter in assessing the validity of the conclusions.

Regardless of the particular line of evidence being considered, certain concepts and constraints affect the interpretation. In particular, there is disagreement over whether an effect, especially one that is reversible or can be compensated, constitutes a hazard. Furthermore, only rarely is any particular study subjected to confirmation by the performance of an identical experiment by another investigator. More often an analogous--but not identical--experiment is conducted with the objective of clarifying or expanding the results of the initial experiment. The second experiment ideally provides a better means of incorporating the findings into the theory that underlies the body of knowledge in a particular field of investigation, but it does not necessarily confirm the results of the first investigation.

Still another consideration is also important: scientific findings are probabilistic in nature, in that facts are known only to some level of probability for a given population; the applicability of those facts to a particular individual may be constrained. For example, the term "median effective dose" for a certain agent refers to the dose that will elicit the response characteristic of that agent in one-half of the exposed individuals. Before the dose is administered, however, one cannot predict whether any specific individual will respond, although the prediction that an individual will have a 50% chance of showing the response is valid. In effect, the probabilistic nature of scientific evidence means that no amount of scientific data can guarantee the absolute safety of any agent for any individual or group of individuals. There is disagreement over whether the conventional scientific approach, whereby an investigator finds or fails to find a statistically significant (very low probability of chance occurrence)

difference between experimental and control groups, is appropriate to considering potential hazards to humans. The scientist's statement that no statistically significant differences between the groups are discernible is not equivalent to the absolute statement that there is no difference between the groups.

Conceivably, agents may have effects that are biologically real but so small in magnitude that the difference in mean response between experimental and control populations may not be discernible within the scattering of values for both populations if the sample sizes are small. Biological studies to detect such small differences and to show that they are statistically significant (to a prespecified probability that they are not due to chance) would require the use of large numbers of animals and, in some cases, long exposure times. The expenditures in time and money necessary to perform such studies may be so large that sponsoring institutions with limited budgets often decide that such studies are not cost-effective in terms of the sponsor's overall objectives. A frequent alternative is to predict effects at very low levels by extrapolation from findings at higher levels, on the basis of assumptions about the mathematical relationship between the level (or dose) of the agent and the degree of the effect. Such assumptions are open to challenge, however, and this approach may lead to disagreement over the possible existence of a threshold dose or dose rate below which the agent has no effects.

It must also be remembered that scientists have personal values, goals, and attitudes. It has been said that there is no such thing as an unbiased expert because becoming an accepted authority involves a personal commitment over a period of time that leads to emphasis of certain viewpoints. Thus, like probabilistic scientific findings, objectivity may well be characteristic of scientists as a group without necessarily being characteristic of any individual scientist. Personal bias can consciously or unconsciously affect how the experiment is designed, how the data are interpreted, and particularly, how the results are applied to decision making. The last is especially important when the decision to be made is in an area outside the scientist's field of expertise.

Finally, scientific experiments are usually restricted to the evaluation of only one factor. In the real world, however, interactions are far more complex. The effect of combinations of factors is illustrated in the incidence of lung cancer in uranium miners, which is higher than in the general population, presumably as a result of the inhalation of radioactive material. The extent of the increased incidence in nonsmoking miners is marginal, but miners who smoke cigarettes have a much higher incidence of lung cancer than either nonsmoking miners or the general population. Thus, scientific evidence can only supply probabilistic information that is relatively narrow in its application to the real world.

C.4 Other Reviews

This section contains descriptions of representative general reviews of the literature on the bioeffects of RFR, including two by Eastern European authors (Baranski and Czerski, 1976; Sudakov and Antimoniy, 1973) and two of Eastern European research by an American (McRee, 1979, 1980). The bibliographies in these reviews served as additional sources of possibly relevant literature citations, thereby ensuring adequate coverage of the literature. Although the conclusions and opinions of the authors of these reviews were carefully examined, the conclusions stated in this assessment of the OTH-B were derived independently from analyses of the primary research literature on each bioeffects topic.

Two useful recent compendia are the issue of the Bulletin of the New York Academy of Medicine (1979) that covers the "Symposium on Health Aspects of Nonionizing Radiation" held at the Academy in April 1979 and the Proceedings of the IEEE, Special Issue on Biological Effects and Medical Applications of Electromagnetic Energy (1980). Both publications contain reviews of specific RFR bioeffects topics as well as reviews of the entire field. The presentations in the Bulletin are directed primarily toward informing physicians about the status of the field, whereas those in the Proceedings are primarily for the nonspecialist in RFR bioeffects. In addition, the Proceedings contains first publication of some recent research results. Only the general review articles in these issues and those selected from earlier publications are considered in this section.

In the Proceedings, Michaelson (1980) presents an overview that includes brief discussions of principles of biological experimentation and interpretation of results, the necessity for and the problems associated with scaling and extrapolating results with animals to effects on humans, and some basic physiological considerations involved in exposure to RFR. He then summarizes the current state of research on all the major bioeffects. He concludes that most of the experimental data indicate that the reported effects of RFR exposure are primarily due to temperature increases or internal changes in temperature gradients, but he recommends further research to resolve substantial uncertainties in certain areas, particularly the effects of chronic exposure to low levels of RFR. Michaelson cites 99 references. An earlier review by Michaelson (1978) covers much of the same subject matter and provides 209 reference citations.

In both the Proceedings and the Bulletin, McRee (1979, 1980) reviews the difficulties in assessing the Eastern European literature on bioeffects of RFR before about 1972, and he discusses the inception of the cooperative agreements between the USSR and the United States. He also indicates that the initial stages of the cooperative program primarily involved exchanges of information (and interchange visits). It became evident that most of the USSR research involved chronic

exposures to average power densities of about 500 microwatts/cm^2 or less, whereas the U.S. research involved relatively short exposures to about 5,000 microwatts/cm^2 or more. This situation led to an agreement to perform duplicate experiments in the two countries.

In the duplicate experiment that McRee describes, rats were exposed at 500 microwatts/cm^2 for 7 hours/day, 7 days/week for 3 months, and specific behavioral and biochemical tests were performed. The U.S. study found a decrease in sulfhydryl activity and blood cholinesterase, as did the USSR study, and blood chemical analyses at the end of the 3-month exposure period showed aldosteronism in the exposed animals, relative to controls, due to vacuolation and hypertrophy in the zona glomerulosa of the adrenal glands. Also, the results showed significant differences in the same direction as those found in the USSR in all behavioral parameters studied (increased threshold in footshock detection, decreased activity in an open field, and poor retention of an avoidance response).

In both reviews, McRee also summarizes effects on humans and animals reported by Soviet, Polish, and Czechoslovakian scientists, and he discusses the safety standards in these and Western countries in the Bulletin. McRee cites 33 references in the Proceedings and 5 references in the Bulletin.

In the Bulletin, Cleary (1979) presents a brief overview of research, with emphasis on reported effects of exposure at low average power densities. He indicates the difficulties in making quantitative comparisons of results and extrapolating from data on animals to effects on humans. He cites 22 references.

In a more comprehensive, earlier review, Cleary (1977) analyzes the results of 12 studies on various aspects of RFR bioeffects and includes references to 100 other articles. He discusses the physical characteristics of RFR, the mechanisms of interaction of RFR with biological systems, and whole-body dose rates and dose-rate distributions within actual and model biological systems. He also reviews the major physiological and behavioral effects of RFR.

Assenheim et al. (1979) provide a report intended for people without scientific backgrounds. It includes discussion of the physical principles involved in the mechanisms of interaction between RFR and biological tissues, applications of RFR (including a brief discussion of OTH radars), treatments of the various RFR-bioeffects topics, comparisons of the exposure standards of various countries, appendices related to specific subjects, a list of the 299 references cited, and a glossary of terms and abbreviations used.

Two reviews, one covering RFR biophysics and the other discussing biological and pathophysiological effects of exposure to RFR, are presented in the transactions of a short course held in Ottawa, Canada,

in June 1978. Lin (1978) presents an assessment of the current knowledge about RFR interactions with biological systems, with emphasis on the dielectric properties of tissue materials, propagation and absorption of RFR in tissues, and basic physical mechanisms of interaction. He cites 76 references.

Stuchly (1977) reviews potentially hazardous RFR emitters, citing 38 references. The review discusses those emitters judged to have potential for producing hazardous levels of RFR under normal operating conditions and under possible malfunction, and considers satellite communication systems and microwave-power devices for generating heat.

Carpenter (1977) gives a critical, comprehensive review of RFR and its effects, emphasizing RFR as an environmental agent. Sections deal with physical characteristics and properties of RFR, effects on tissue, "thermal" and "nonthermal" effects, exposure levels, biological effects of RFR on human beings and experimental animals, and RFR effects on the eye, the testes, and the nervous system, and on development. Carpenter cites 110 references.

Dodge and Glaser (1977) assess international trends in research, development, and occupational health and safety, concentrating on events since 1975. Some 25 references are cited. Sections discuss exposure standards, research on bioeffects, effects of RFR on humans, and U.S. federal RFR health and safety programs.

The 234-page book by Baranski and Czerski (1976), published in English (translation by Czerski), is a comprehensive Eastern European presentation of the then current literature and research results through 1975. The book contains references to 614 articles; Western, as well as Eastern European, investigations are well represented. The seven chapter headings are:

- Introduction
- Physical Characteristics of Microwaves
- Interaction of Microwaves with Living Systems
- Biological Effects of Microwaves. Experimental Data
- Health Status of Personnel Occupationally Exposed to Microwaves, Symptoms of Microwave Overexposure
- Safe Exposure Limits and Prevention of Health Hazards
- Final Comments

Sudakov and Antimoniy (1973) provide an extensive review (224 references) of the neurophysiology and behavior of animals and humans in an English translation of the original Russian article by the Joint Publications Research Service. The authors appear to accept as uncontestable the premise that RFR has direct effects (denoted by them as "nonthermal") on the nervous system of animals. The review is in two main sections. The first concerns biological aspects of the effects of RFR on the central nervous system (CNS) of animals and humans; it

contains subsections on natural RFR as a factor in evolution, the sensing of RFR by living organisms, and the effects of natural RFR on animals and humans, on the activity of the CNS, and on the behavior and conditioned activity of animals and humans. The second main section concerns neurophysiological mechanisms of the action of RFR, with subsections on bioelectrical activity of the brain during exposure to RFR, morphological and functional changes in the CNS on exposure to RFR, and selective action of RFR on structures of the CNS.

C.5 Present State of Knowledge Regarding Physical Effects

C.5.1 Interactions of RFR with Biological Entities

Interactions of electromagnetic fields with biological entities are often loosely characterized in the bioeffects literature as "thermal" or "nonthermal," a usage that has led to confusion and controversy. Therefore, it is appropriate at this point to introduce working definitions of these terms, with the recognition that the boundary between these types of interaction is not sharp.

The interaction of an agent (e.g., RFR) with an entity (biological or nonbiological) can be characterized as thermal if the energy absorbed by the entity is transformed at the absorption site into heat. Heat absorption, in turn, is defined in classical thermodynamics as either an increase in the mean random speed (or kinetic energy) of the molecules at the site (a local increase in temperature), or as an increase in the disorder or randomness of the molecular motion without an increase in mean random speed (a first-order phase change, such as the process involved in ice melting at 0 deg C), or both.

An entity can also absorb energy at specific discrete frequencies in the form of energy packets or "quanta," each of which has an energy proportional to one of the discrete frequencies. Although large numbers of molecules can be involved, quantum absorption is essentially a microscopic phenomenon in that the constituents and configurations of the various molecular species comprising the entity determine the specific frequencies or characteristic spectra at which such absorption can occur. The kinds of interactions involved are numerous and of varying degrees of complexity. They include alterations of molecular orientations and configurations that do not change the basic identities of the molecules, disruption of intermolecular or intramolecular bonds, and excitation of atoms or molecules to higher electron states (including ionization). Such interactions can be characterized as "short-range" processes.

Cooperative interactions also occur among subunits of molecules within biological cells, in cell membranes, and in extracellular fluids. Cooperative interactions are often characterized as "long-range" because absorption of energy at one specific site in a structure, e.g., in a membrane or in a biological macromolecule, can

affect a process elsewhere in the structure, or a function of the structure as a whole can be triggered by the release of energy stored in the structure, thereby producing biological amplification.

Conceptually, all such quantum interactions can be characterized as "nonthermal." However, if most of the energy thus absorbed is subsequently transformed locally into heat (as defined above), the distinction between nonthermal and thermal is blurred. Pragmatically, therefore, characterization of an interaction of RFR with a biological entity as nonthermal requires that the interaction give rise to a frequency-specific effect that is experimentally distinguishable from heating effects caused by thermalization of the absorbed RFR energy.

C.5.1.1 Thermal Interactions

Consider now the incidence of continuous-wave (CW) RFR on a human or an animal. The relative magnetic permeability of most organic constituents is about unity. Therefore, thermal interactions (as defined above) can be described in terms of the dielectric, electrical-conductivity, and thermal properties of the body organs, tissues, fluids, and so forth, as well as the characteristics of the RFR (frequency, power density, polarization). Measurements of these properties have been made for various mammalian tissues, blood, cellular suspensions, protein molecules, and bacteria over the spectral region from about 10 Hz to 20 GHz, notably by Schwan and coworkers (Schwan and Foster, 1980; Schwan, 1963, 1957; Schwan and Piersol, 1955; Schwan and Li, 1953) and others (Lin, 1975; Cook, 1951, 1952). In general, the dielectric constants were found to vary inversely with frequency in a separate characteristic manner for each of three parts of that frequency range (the "alpha," "beta," and "gamma" dispersion regions), as shown for muscle tissue in Figure C-2. These dispersion regions are ascribed to different predominant relaxation mechanisms, each characterized by specific time constants (Schwan, 1957). In the low and intermediate frequency ranges (about 10 Hz to about 100 MHz), which encompass the "alpha- and beta-dispersion" regions, the properties of cell membranes, which have large specific capacitances (about 1 microfarad/cm^2), predominate. In the range above about 10 GHz (the "gamma-dispersion" region), membrane impedances are negligible, and the behavior of the water and electrolyte content are most predominant. As an example of the large numerical variation of dielectric constant, the values for muscular tissue decrease by five orders of magnitude, from about 3 x 10^6, at 10 Hz to 30 at 20 GHz.

In the frequency range from 3 to 30 MHz, the dielectric constant of muscle varies from about 360 to about 110. The values for skin, blood, and other tissues with high water contents are comparable. The values for fat, bone, and other tissues with low water contents are about an order of magnitude smaller and are sensitive to the amount of water the tissues contain.

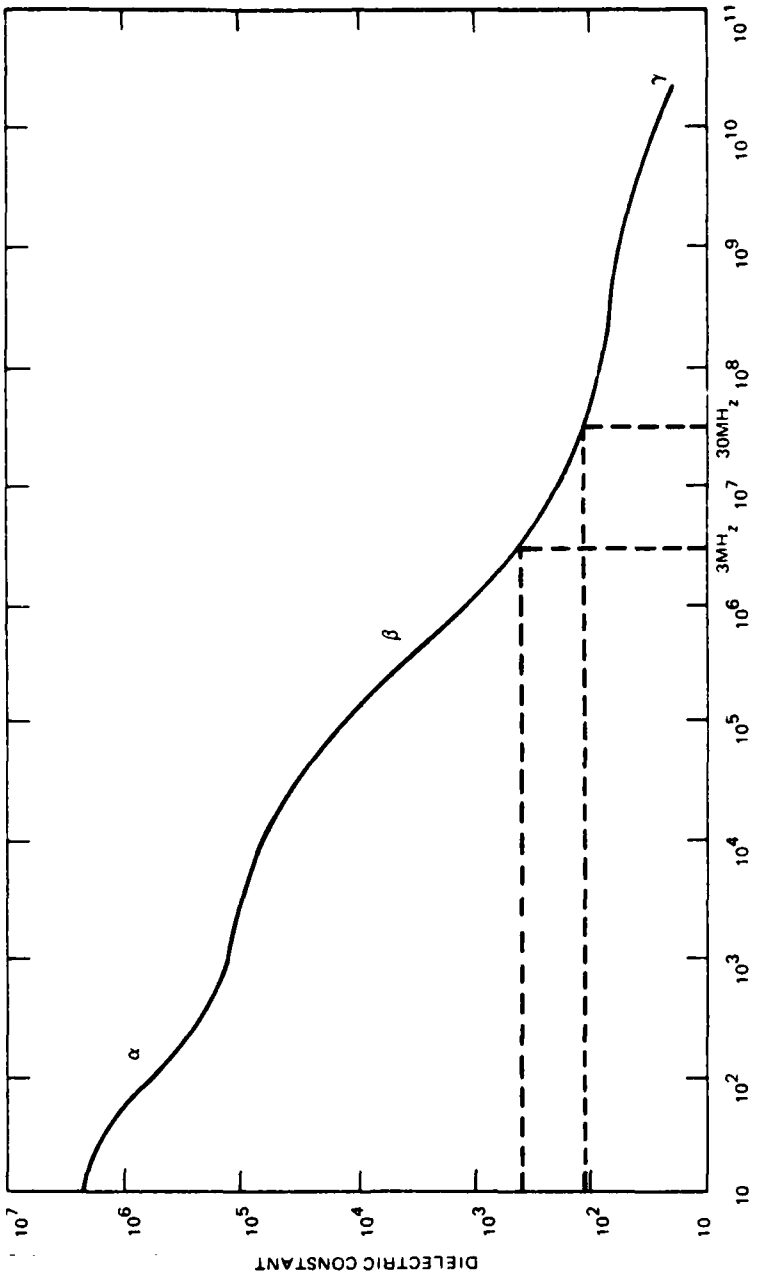

FIGURE C-2 DIELECTRIC DISPERSION FOR MUSCLE TISSUE

Because the index of refraction of any material is related to its dielectric constant, electromagnetic fields are reflected and refracted at the air-surface interface and at internal boundaries between constituents of widely different dielectric properties--e.g., at interfaces between the skull and the dura or between a body cavity and adjacent tissues--thereby affecting the internal field distributions. In the 3 to 30 MHz range, for example, about 95% to 85% of the incident power density is reflected at the air-skin interface (Johnson and Guy, 1972), and the approximately 5 to 15% that enters the body is progressively attenuated with depth because of energy absorption in the tissues.

The attenuation constant (rate of energy absorption with distance) of any material is proportional to the square root of its electrical conductivity. The concept of "penetration depth" (the inverse of attenuation constant) is often used. For homogeneous specimens, the penetration depth is defined as the distance at which the electric field amplitude is 1/e (37%) of its value or the power density is $1/e^2$ (14%) of its value just within the surface. The electrical conductivities of skin, muscle, blood, and other constituents of the body increase slowly with frequency up to about 1 GHz and rapidly from about 1 GHz upward. The penetration depths for skin, muscle, and other high-water tissues are about 91, 22, and 14 cm at 1, 10, and 27 MHz, respectively; the values are about an order of magnitude greater for fat. (At about 10 GHz and higher, field penetration is largely confined to the skin.) Thus, although RFR at the frequencies in the OTH-B range is deeply penetrating, most of the incident power density is reflected at the surface of the body.

C.5.1.2 Dose-Rate Considerations

In the literature on bioeffects of RFR, thermal energy absorption from an electromagnetic field is usually characterized by the Specific Absorption Rate (SAR), which is defined as the rate of energy absorption per unit volume in a small volume at any locale within an entity, divided by the mean density of the constituents in that volume. SAR is expressed in terms of W/kg or mW/g (1 mW/g = 1 W/kg). The numerical value of SAR in any small region within a biological entity depends on the characteristics of the incident field (power density, frequency, polarization), as well as on the properties of the entity and the location of the region. For biological entities that have complex shapes and internal distributions of constituents, spatial variations of SAR cannot readily be calculated. Therefore, the concept of "whole-body SAR," which represents the spatial average value for the body per unit of incident power density, is often used because it is a quantity that can be measured experimentally--e.g., by calorimetry--without information on the internal SAR distribution.

Many investigators have studied relatively simple geometric models, including homogeneous and multilayered spheroids, ellipsoids, and

cylinders that have weights and dimensions approximately representative of various species, including humans. For studies pertaining to the far fields of RFR sources, such models were actually, or were assumed to be, irradiated with linearly polarized plane waves to determine the dependence of whole-body SAR on frequency and orientation relative to the polarization direction of the RFR. Many of the significant data have been included in a series of handbooks (Durney et al., 1980, 1978; Johnson et al., 1976) that are useful for very approximate frequency-scaling and interspecies comparisons of whole-body SAR values. An important result of this work is that the largest value of whole-body SAR is obtained when the longest dimension of each kind of model is parallel to the electric component of the field and when the wavelength of the incident RFR is about 2.5 times the longest dimension. The adjective "resonant" is often applied to the frequency corresponding to this wavelength. The resonant value of whole-body SAR for each model is also inversely dependent on the dimension perpendicular to the polarization direction (and propagation direction) of the field; i.e., the model has characteristics somewhat similar to those of a lossy dipole antenna in free space. Resonances would also occur for circularly polarized RFR. Such RFR can be resolved into two mutually perpendicular components, each having half the total power density. Therefore, an entity exposed to circularly polarized RFR would have lower resonant SAR values than it would have if exposed to linearly polarized RFR of the same total power density.

Figure C-3 shows plots of whole-body SAR versus frequency for a prolate-spheroidal model of an "average" or "standard" man, approximately 5 ft 9 in. (1.75 m) tall and weighing about 154 lb (70 kg), exposed to 1 mW/cm^2 of plane-polarized RFR in three orientations relative to the polarization direction. A relatively sharp peak is obtained at resonance for the "E" orientation in which the long axis of the prolate spheroid is parallel to the polarization direction (electric vector) and perpendicular to the magnetic vector and propagation direction. (In the "H" orientation, the long axis is parallel to the magnetic vector and perpendicular to the electric vector and propagation direction; in the "K" orientation, the long axis is parallel to the propagation direction.) For this model of man, the resonant frequency (in the E orientation) is about 70 MHz; at this frequency, the SAR is about 0.2 W/kg for 1 mW/cm^2 incident power density, or about 1/6 of his resting metabolic rate or about 1/21 to 1/90 of his metabolic rate when performing exercise ranging from walking to sprinting (Ruch and Patton, 1973). Similarly, the resonant frequency for an "average" woman about 5 ft 3 in. tall is about 80 MHz, and her whole-body SAR is about the same as for the average man. The resonant frequency for a 10-year-old is about 95 MHz; for a 5-year-old, about 110 MHz; and for a 1-year-old, about 190 MHz. The resonant SAR values for such children are about 0.3 W/kg for 1 mW/cm^2.

Below resonance in the E orientation, the whole-body SAR is approximately proportional to f^2; above resonance, the SAR is

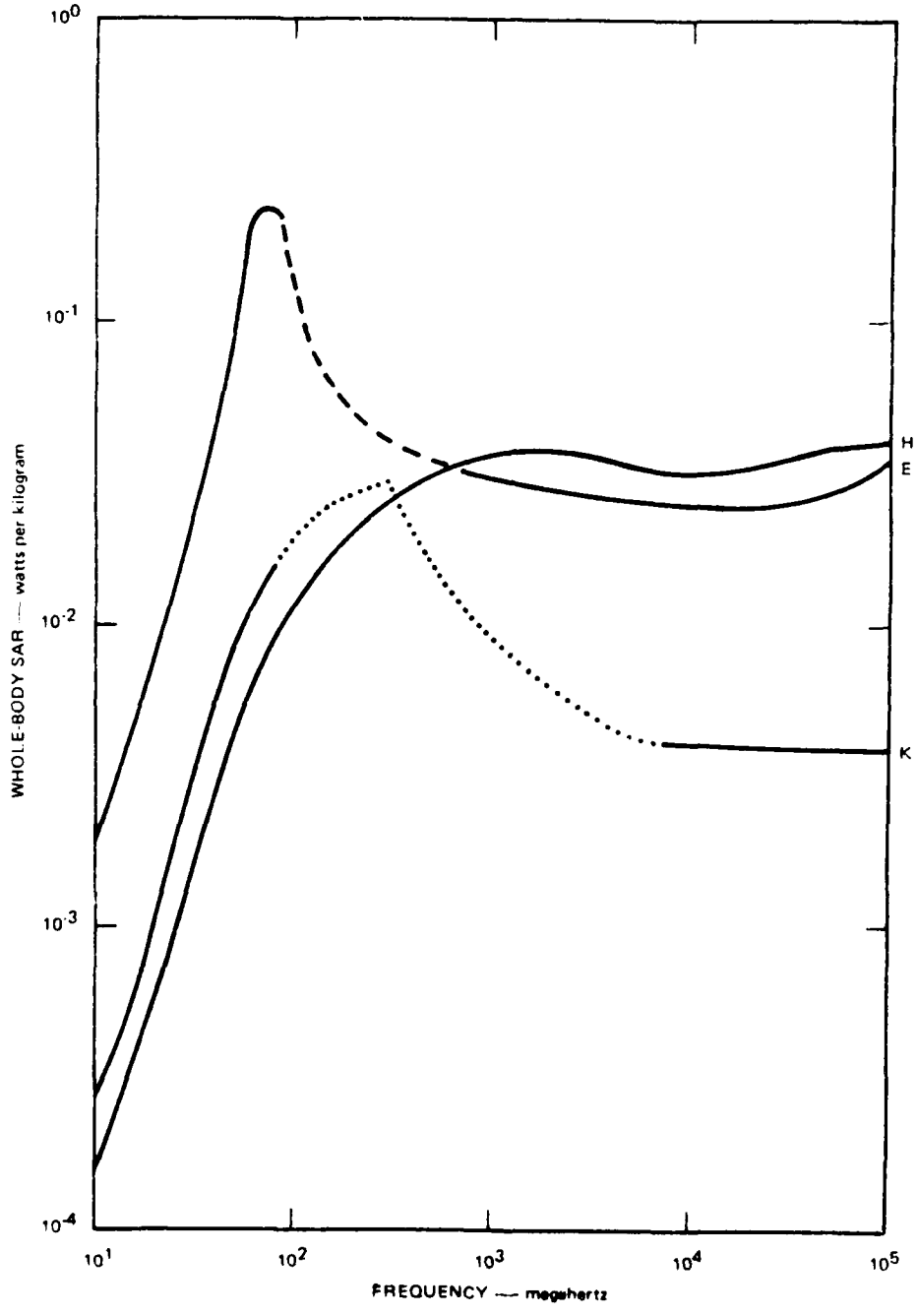

FIGURE C-3 WHOLE-BODY SAR FOR PROLATE-SPHEROIDAL MODEL OF "AVERAGE" MAN EXPOSED TO 1 MILLIWATT PER SQUARE CENTIMETER OF RADIOFREQUENCY RADIATION

approximately proportional to 1/f for about one decade of frequency, and it levels off thereafter.

The foregoing discussion of whole-body SAR is also largely applicable to modulated RFR (including pulsed RFR) at corresponding carrier frequencies and time-averaged incident power densities.

To illustrate how the concept of whole-body SAR could be interpreted, consider the standard model man. Absorption of energy as heat by exposure of such a model man at his resonant frequency (70 MHz) in the E orientation to an average power density of 1 mW/cm^2 (SAR of 0.2 W/kg) for 1 hour would produce a mean body temperature rise of only about 0.2 deg C if no heat removal mechanisms are present and if no first-order phase changes are involved. At 30 MHz, the SAR is about 0.02 W/kg per mW/cm^2 for the E orientation (and much lower for the H and K orientations), as seen in Figure C-3. Therefore, exposure of the model man to the same power density for the same duration, but at 30 MHz, would produce a mean temperature rise of only about 0.02 deg C.

Figure C-4 presents similar data for a prolate-spheroidal model of a "medium" rat (0.2 m long and weighing 0.32 kg). Not only is the resonant frequency (approximately 650 MHz) higher than any of the values for humans, but the resonant SAR is also larger (about 0.8 W/kg for the rat, compared with about 0.2 for man, per mW/cm^2 of incident power density). Therefore, scaling of data from experimental animals to humans must consider such differences of whole-body SAR as well as frequency. To illustrate this point, the SAR of the medium rat is about 0.2 W/kg per mW/cm^2 (in either the E or H orientation) at 2.45 GHz, a frequency that has been used in many laboratory studies of bioeffects of RFR. Coincidentally, this SAR value is about the same as that for the average man at resonance, but it is 10 times as great as his value at 30 MHz. Therefore, extrapolation of any bioeffects seen in rats from exposure to 1 mW/cm^2 at 2.45 GHz to humans exposed to the same power density at 30 MHz (or lower) would be highly questionable. Alternatively, to obtain the same SAR in the medium rat as that for the average man exposed to any given value of power density at 30 MHz (e.g., 1 mW/cm^2) it would be necessary to expose the rat to one-tenth of that power density (i.e., 100 microwatts/cm^2 in this numerical example) at 2.45 GHz.

Numerical calculations of internal spatial distributions of SAR have been performed on "block" models, in which the shape of the body is approximated by an appropriate arrangement of many cubical cells, with each cell assumed to be biologically homogeneous and to have constant internal field over its volume when the model is exposed to RFR (Hagmann et al., 1979a). In addition, more accurate values of whole-body SAR have been obtained from such spatial distributions than from simpler models.

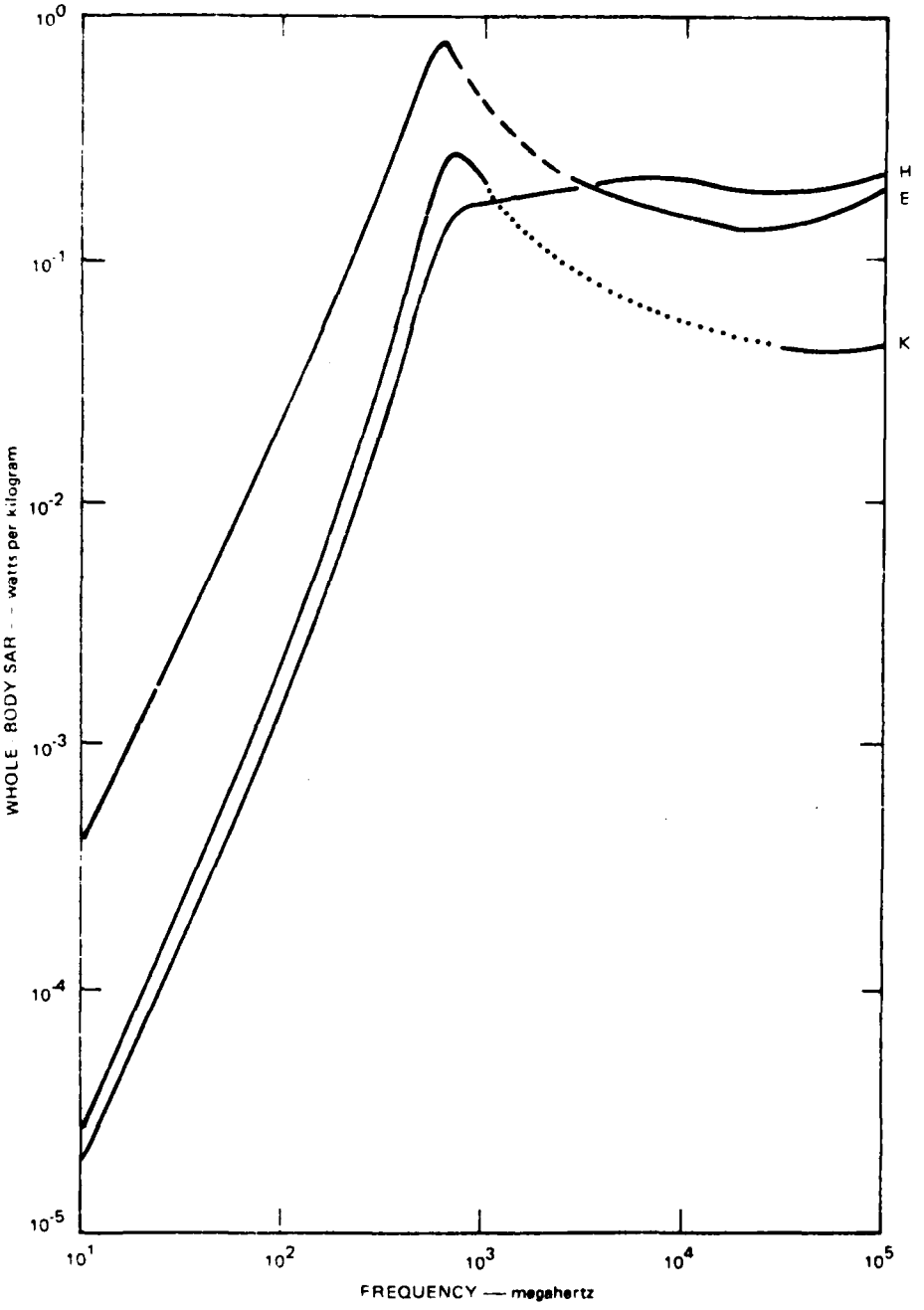

FIGURE C-4 WHOLE-BODY SAR FOR PROLATE-SPHEROIDAL MODEL OF "MEDIUM" RAT EXPOSED TO 1 MILLIWATT PER SQUARE CENTIMETER OF RADIOFREQUENCY RADIATION

The presence of a ground plane or other reflecting surfaces shifts the resonant frequencies downward and can produce higher values of whole-body SAR at the lower resonant frequencies (Hagmann and Gandhi, 1979; Gandhi et al., 1977; Gandhi, 1975). Especially pertinent to OTH-B are the results of Hagmann and Gandhi (1979). Their calculations show that for a homogeneous block model of the average or "standard" man in electrical contact with an infinite, perfectly conducting ground plane, the whole-body resonant frequency (in the E orientation) is shifted from the free-space value of 77 MHz to 47 MHz; i.e., to a value much closer to the OTH-B frequency band. Moreover, the whole-body SAR at 47 MHz is 32.5% higher than at 77 MHz. However, they also note that such ground-plane effects are largely eliminated if conductive contact with the ground is removed.

Block models as well as homogeneous and multilayered spheroidal and cylindrical models having appropriate electromagnetic and thermal characteristics have also been used to represent various parts of the body, such as the head and limbs (Hagmann et al., 1979a, 1979b; Massoudi et al., 1979; Rukspollmuang and Chen, 1979; Wu and Lin, 1977; Neuder et al., 1976; Kritikos and Schwan, 1975, 1976; Lin, 1975; Weil, 1975; Joines and Spiegel, 1974).

An early, very significant finding for spherical models of the isolated head assumed to be exposed to plane-wave RFR was the discovery of local regions of relative maximum SAR values. The locations of such regions depend on the size of the head, the electromagnetic characteristics of its layers, and the wavelength of the incident field. These regions have been conveniently dubbed "hot spots," even for combinations of incident power density and exposure duration that would produce biologically insignificant temperature increases at such spots. An analysis of a homogeneous lossy spherical head model (Kritikos and Schwan, 1975) indicates that there are hot spots inside spheres having radii between 0.1 and 8 cm, and in the frequency range from about 300 MHz to 12 GHz; for larger radii and other frequencies, there are internal hot spots, but the hottest spots are at the front surface (facing the RFR source). Similar results were obtained for multilayered spherical models (Kritikos and Schwan, 1976; Weil, 1975). Specifically, Kritikos and Schwan (1976) analyzed two such models, one with a radius of 5 cm and the other, 10 cm. For the 5-cm head, the hot spots are internal over the frequency range from about 400 MHz to 3 GHz. The highest relative maximum SAR occurs near the center of the head at a frequency of about 1 GHz, and has a value of about 9 W/kg for an incident power density of 1 mW/cm^2. (Of course, the whole-head SARs are considerably lower.) By contrast, for the head of 10-cm radius (about that for an adult human head), no deep internal hot spots are produced at any frequency; the hot spots are always at or just beneath the front surface.

Rukspollmuang and Chen (1979), starting with a block model of a multilayered isolated spherical head, found qualitatively similar

results. They then studied a block model having a shape and internal structure more closely approximating that of the human head (including eyes, nose, skull bone, and brain) at 918 MHz and 2.45 GHz, and found that much of the energy within the head would be absorbed by the skull. Specifically, for frontal exposure of this model at 918 MHz, the maximum SAR for the brain region is about one-third that for the brain region of a 7-cm-radius multilayered spherical model. Also, for frontal exposure of the more accurate model to 2.45 GHz, the induced field is concentrated primarily near the proximal surface, and therefore energy dissipation within the brain would be relatively low.

Hagmann et al. (1979b) calculated SAR distributions in the attached head of a block model of the human and derived whole-head as well as whole-body SARs for three orientations of the model relative to the RFR source. For front-to-back propagation with the long axis of the body parallel to the electric vector, they found a rather broad head resonance at about 350 MHz with a whole-head SAR of 0.12 W/kg per mW/cm^2; the corresponding whole-body SAR is about 0.05 W/kg. A sharper head resonance at 375 MHz was obtained for head-to-toe propagation, with whole-head and whole-body SARs of 0.22 and about 0.07 W/kg per mW/cm^2, respectively.

Such analyses of isolated- and attached-head SARs indicate that thermal brain damage is most unlikely to occur in the human head from RFR in the 3-30 MHz range unless the incident power density and exposure duration are sufficient to heat the entire head to temperatures substantially greater than normal.

Results of theoretical analyses of SARs have been verified experimentally. Physical models of simple geometry or in human- or animal-figurine shape have been constructed from synthetic biological materials that have approximately the same electromagnetic characteristics as their corresponding biological constituents; the models were then exposed to sufficient power densities to obtain readily measurable temperature increases, which were measured immediately after irradiation. To use available sources of RFR that provide only specific frequencies, and to avoid the problems of exposing large full-scale models, models of smaller size are often chosen by the use of scaling relationships so as to permit extrapolation of experimental results on such smaller models exposed at the available frequencies to obtain results on full-size models at other frequencies of interest. This approach was taken by Guy et al. (1976), who exposed homogeneous human figurines having lengths of 37.6 and 26.5 cm (as well as spheres and ellipsoids) at approximately 143 MHz to simulate exposures of full-size figurines (1.74 m in length) at 31.0 and 24.1 MHz. In their study of head resonances, Hagmann et al. (1979b) exposed human figurines with lengths of 20.3, 25.4, 33.0, and 40.6 cm at 2.45 GHz, to correspond with full-size figurines exposed at scaled frequencies of 284.5, 355.6, 462.3, and 569.0 MHz, respectively.

A technique widely used to determine SAR distributions in physical models or actual animal carcasses is to embed the objects in styrofoam, section the experimental object along parting planes of interest, then reassemble the object and expose it to RFR. The spatial distribution over each parting plane is measured with scanning infrared thermography immediately after exposure. However, such spatial temperature distributions should not be regarded as corresponding with in vivo internal temperature distributions, because the heat-transfer characteristics of such carcasses and physical models are significantly different from those of live animals, and do not have the thermoregulatory mechanisms of the latter. Instead, such measured temperature distributions represent approximations to the internal field or SAR distributions.

Among the interesting results obtained by Guy et al. (1976) were that exposure of a full-size human figurine to an electric field parallel to its length at frequencies in the HF band yields relatively high SARs in regions of the body where the cross-section perpendicular to current flow is relatively small, such as the neck, knees, and ankles. Also, exposure of the figurine to a magnetic field perpendicular to the frontal plane in the same frequency range produces eddy currents that yield relatively high SARs where such currents are forced through relatively small cross-sectional areas or are diverted by sharp angular changes, such as in the groin and along the sides of the body near the ribs. These results are especially pertinent to near-field exposure situations, for which it is necessary to measure the spatial variations of the electric and magnetic fields separately because their amplitude ratio may vary from point to point and they may not be perpendicular to each other and to the propagation direction.

Calorimetry was used to measure whole-body (and whole-head) SARs by Hagmann et al. (1979c) and by others (Allen and Hurt, 1979; Kinn, 1977; Hunt and Phillips, 1972). Whole-body SARs were also determined in waveguide types of exposure systems by measuring the input, output, and reflected values of RFR power without and with the object present and performing the requisite arithmetic (Ho et al., 1973).

On the basis of such theoretical and experimental SAR considerations, it seems unlikely that chronic exposure of humans (in vivo) to the RFR from OTH-B at the average power densities outside the exclusion fence at ground levels will cause any significant increase in local internal or mean body temperature.

C.5.1.3 Quantum Interactions

Regarding quantum interactions of CW RFR, the activation energies for short-range effects at the molecular level extend from about 0.08 eV (1.3×10^{-20} J) for hydrogen-bond disruption to about 10 eV (1.6×10^{-18} J) for ionization. The corresponding quantum frequencies range from about 19 to 240 THz (Cleary, 1973). However, an electromagnetic

quantum at 30 MHz has an energy of only 1.2×10^{-7} eV (1.9×10^{-26} J), or approximately one-millionth of the energy required for hydrogen-bond disruption, which is at the lower end of the energy-activation range cited above. Therefore, the existence of nonthermal biological effects of CW RFR ascribable to such short-range molecular interaction mechanisms is extremely doubtful.

Biological generation of fields having frequencies in the range below 100 Hz, such as those measured by the electroencephalogram (EEG), is regarded as evidence for the occurrence of cooperative or long-range interactions. Several theoretical models of neuronal membranes (e.g., Schmitt and Samson, 1969; Frohlich, 1975a; Frohlich, 1975b; Grodsky, 1976) indicate that activation energies or frequencies for cooperative processes can be much lower than those for short-range interactions. Because the thermal energy corresponding to the physiological temperature 37 deg C is about 0.027 eV, corresponding to a spectrum that encompasses the quantum frequency range for cooperative processes, the question has been raised whether postulated effects of weak RFR on cooperative processes, based on theoretical models, would be distinguishable from effects that are spontaneously induced thermally. Alternatively, separation of such RFR interactions from those thermally induced may require that the rates of occurrence of the former exceed the rates for the latter. This requirement implies that for manifestation of such effects of RFR, the intensity of the incident field must exceed minimum values or thresholds related to the specific processes.

Because predictions from various theoretical models and related considerations conflict to a significant extent (see Adey and Bawin, 1977; Taylor and Cheung, 1978), the issue of whether weak external fields at frequencies well below the infrared range (i.e., RFR) can alter biological processes is not yet resolved. However, effects ascribed to such cooperative processes have been reported, notably field-induced increases and decreases of calcium-ion binding to cell membranes, a phenomenon called "calcium efflux." This phenomenon is discussed in Section C.6.5.2.

C.5.1.4 Interactions of Modulated RFR

Insofar as average power density is concerned, the effects of amplitude-modulated RFR at any given carrier frequency and power density are essentially the same as those of CW or frequency-modulated-CW (FMCW) RFR at the same carrier frequency and power density. In addition, biological effects have been ascribed to amplitude modulation per se, notably the previously mentioned calcium-efflux phenomenon, which was seen for 147-MHz and 450-MHz RFR modulated at sub-ELF frequencies, but not for unmodulated RFR at these carrier frequencies. However, the calcium-efflux phenomenon is not of concern for OTH-B because the RFR therefrom is FMCW. Also, there is no evidence that frequency modulation causes any biological effects ascribable specifically to the modulation.

C.5.1.5 Interactions of Pulsed RFR

Although the RFR from OTH-B is not pulsed, the interactions of high pulse-power-density RFR at low average power densities are discussed herein because such interactions are often cited as being "nonthermal" effects.

The temperature increase of any given region within a biological entity due to the arrival of a single RFR pulse would be small, because of the relatively large thermal time constants of biological materials and the operation of heat-exchange mechanisms. However, if the region contains a boundary between layers of widely different dielectric properties, then the temperature gradient (rate of change of temperature with distance) can be large at such a boundary even though the mean temperature increase in the region is small.

One single-pulse effect known to occur in vivo is the phenomenon of "microwave hearing" (Frey and Coren, 1979; Frey, 1961) discussed in Section C.6.5.1, or the perception of single or repetitive short pulses of RFR as apparently audible clicks. The interaction mechanisms involved are not yet completely understood. However, most of the experimental results tend to support the theory that pulse perception occurs because of transduction of the electromagnetic energy into sound pressure waves in the head at a boundary between layers having widely different dielectric properties (e.g., at the boundary between the skull and the skin or dura). The energy in a pulse arriving at such a boundary is converted into an abrupt increase in momentum that is locally thermalized, producing a negligible volumetric temperature increase but a large temperature gradient across the boundary. Under such conditions, rapid local differential expansion would occur and create a pressure (sound) wave that is detected by the auditory apparatus. This effect is often characterized as nonthermal because the power density averaged over two or more pulses can be miniscule. Specifically, the time-averaged power density for two successive pulses is inversely proportional to the time interval between the arrival of the pulses at the perceiver, and this interval can be indefinitely long without affecting the perception of each pulse. Therefore, the time-averaged power density has no relevance to perception. Irrespective of how the RFR-hearing phenomenon is characterized, the significant point is that the preponderance of experimental evidence indicates that the pulses are converted into actual sound in the head, rather than perceived by direct RFR stimulation of the auditory nerves or the brain.

As discussed in Sections C.6.5.3 and C.6.6.1, pulsed RFR has been reported to produce other effects, such as alterations of the blood-brain barrier and behavioral changes. However, neither the auditory effect discussed above nor these other effects are likely to be of concern with regard to the OTH-B, because the RFR therefrom is not pulsed.

C.5.2 Instrumentation for Densitometry and Dosimetry

Much of the early laboratory research on bioeffects of RFR suffered from lack of adequate instrumentation for measuring incident fields or energy absorption rates (e.g., as internal temperature increases at high incident levels) within biological entities. Moreover, the available instrumentation was often incorrectly used, or was the source of significant errors in numerical values, or of spurious biological findings (artifacts) traceable to perturbations introduced by the presence of the sensors. For these reasons, many of the early results should be viewed as questionable, at least from a quantitative standpoint. During recent years, however, major advances have been made in instrumentation, both for determining incident-field intensities for biological research, and for determining internal energy-absorption rates.

Considering first the instrumentation for determining incident fields, a representative device for measuring average power densities is the commercially available (from NARDA Microwave Corp.) broadband isotropic monitor (Aslan, 1972). Its sensors consist of linear arrays of thermocouple elements, each array comprising a lossy antenna of relatively small length and capable of adequate response over the frequency range from 300 MHz to 18 GHz, for which a calibration curve is provided by the manufacturer. Isotropic response is obtained by incorporation of three mutually perpendicular sensor arrays. To minimize errors in the direct-current output values of the sensor assembly caused by possible induction of spurious RF currents in the lead wires, the wires used are of very high resistivity (about 200 kilohms/m or 60 kilohms/ft). Also, the sensors are only lightly coupled to the incident field, so that perturbations of the field caused by scattering are minimal. The sensors respond to the mean-square of only the electric component of the field. Nevertheless, the use of the instrument for measuring average power densities in the far-field region is fully justified because the ratio of the amplitudes of the electric and magnetic components has essentially the same value (377 ohms, the "impedance" of free space) at all points in that region, and the instrument is calibrated to read total average power density. (In the induction and near-field regions of an antenna, it may be necessary to measure the intensities of both the electric and magnetic components.) The most sensitive model of this instrument has a full-scale range of 200 microwatts/cm^2.

A more recently developed instrument (now commercially available from Vitek, Inc.) is the National Bureau of Standards (NBS) Model EDM-2 Electric Energy Density Meter, designed for the 10-to-500 MHz range (Belsher, 1975; Bowman, 1974). Its sensor consists of three mutually perpendicular integral dipole-diodes ("rectennas") that also respond only to the electric component of the field. An 18-inch handle from the sensor contains high-resistivity lead wires to minimize field perturbation and spurious pickup. The most sensitive range of the

instrument is 0.003 microjoules/m^3 full-scale (equivalent to approximately 176 microwatts/cm^2), and its response time (rise time plus fall time) is about 1 ms in this range.

Also now commercially available (from General Microwave Corp.) is an isotropic probe covering the frequency range from 200 KHz to 26 GHz; its highest sensitivity range is 20 microwatts/cm^2 full-scale.

Commercial organizations also market other "radiation hazard" instruments, including meters for checking leakage from microwave ovens.

Field survey instruments of this kind have been analyzed for possible sources of error (Wacker and Bowman, 1971). Because of the relatively long response times of such instruments, they cannot be used for measuring the pulse power densities of short pulses. Therefore, in research programs on possible bioeffects of pulsed fields, incident pulse power densities are usually calculated from measurements of average power density and duty cycle (or pulse duration and pulse repetition frequency) made with commonly available and readily calibrated components and instrumentation.

Magnetic-field probes have been developed for relatively low frequency ranges, as exemplified by the two devices developed at NBS for near-field measurements in the Industrial, Scientific, and Medical (ISM) bands within the range from 10 to 40 MHz (Greene, 1975). The probes consist of single-turn, balanced-loop antennas of 10-cm and 3.16-cm diameter for the amplitude ranges 0.5 to 5 A/m and 5 to 50 A/m, respectively. (The free-space equivalent power density is proportional to the square of the amplitude. For example, the power density equivalents to 0.5 and 5 A/m are approximately 10 and 1,000 mW/cm^2, respectively.)

A probe for simultaneously measuring the electric (E) and magnetic (H) components at the same locale in the near field has been developed for the frequency range from 10 to 100 MHz (Babij and Bassen, 1980). A single-axis probe consisting of an electric dipole suitably mounted within a magnetic loop has been tested. The linear range of the H probe is from 0.016 to 0.37 A/m, and that of the E probe from 10 to 200 V/m. An isotropic probe consisting of three mutually perpendicular single-axis probes has been designed.

Regarding methods for determining whole-body dose rates for biological entities and dose-rate distributions within such entities, calorimetry for the former and scanning infrared thermography for the latter (previously discussed in Section C.5.1.2) continue to be important techniques that are applicable primarily to animal carcasses and physical models of various species constructed from synthetic biological materials. It is important to note that because of differences in heat-transfer characteristics and the absence of thermoregulatory mechanisms, temperature distributions measured within a

carcass by infrared thermography do not represent the in vivo temperature distributions for that animal; rather, they correspond to the internal-field distributions induced by the incident RFR.

Probes have been implanted or inserted to measure local RFR-induced temperature changes or fields within animals during irradiation in vivo, often with the introduction of artifacts. However, recent developments of probes have largely diminished the problem of perturbation of the temperature or local field caused by the presence of the sensor and its lead wires. Such developments have also reduced the size of readout errors caused by pickup of the incident field in the lead wires and by the presence of spurious potentials at junctions between sensors and lead wires. The miniaturized isotropic dipole-diode probe developed by Bassen and co-workers (Bassen et al., 1975, 1977), the liquid-crystal/fiber-optic probe developed by Johnson and co-workers (Johnson et al., 1975), and the nonmetallic thermocouple developed by Olsen and Molina (1979) are representative examples of such progress.

Efforts are also being made to reduce errors and artifacts in measurements of biologically generated fields and potentials--such as the EEG and the electrocardiogram (EKG)--in the presence of the incident RFR. Chou and Guy (1979a) have developed electrodes that can be implanted in the cortex or subcortex for measuring the EEG during chronic exposure to RFR. These electrodes are made of carbon-loaded teflon that has an electrical conductivity close to that of tissue, and they have been shown to be nonpolar, thereby minimizing field perturbations and spurious local potentials. The electrodes have also been shown to have good tissue compatibility by histological examination after 4 to 6 months of implantation. Several high-resistivity electrodes have also been developed by Tyazhelov et al. (1977).

The use of RFR for imaging internal organs is being developed. For example, Larsen and Jacobi (1979) used a pair of waveguide antennas (one for transmitting and the other for receiving) submerged in water to obtain images of the interior of an excised canine kidney with a resolution of about 5 mm. The kidney was suspended between the antennas, and the antennas were slowly moved as a unit perpendicular to the propagation direction in a successive line pattern (raster) relative to the kidney by a stable electromechanical scanning system. The frequency used was 3.9 GHz, which corresponds to a wavelength of about 8.5 mm in water. The use of submerged phased-array antennas to decrease the scanning time is currently under development.

C.6 Present State of Knowledge Regarding Biological Effects

C.6.1 Epidemiology

Epidemiology, as used in the context of this document, refers to studies of whether one or more health-related conditions can be associated statistically with purported or actual exposure of humans to

RFR (in contrast with assessments based on extrapolation from data on animals to humans). Epidemiological results tend to be based on imprecise estimates of exposure characteristics (frequency, power density, and duration). The extent to which the control group matches the exposed group is sometimes open to question. Because matching on all relevant factors except exposure is the basis for concluding that any observed differences between groups are related to the RFR exposure, selection of an appropriate control group is critical. Despite these limitations, such studies do provide almost the only direct information available on possible effects of RFR exposure in humans.

A group of reports was selected for review from the literature in the United States, Poland, Czechoslovakia, and the USSR. These reports provide a representative sample of the kinds of information currently available.

The U.S. Embassy in Moscow has been subjected to RFR since 1953, the year after the United States moved its chancery to Chekovsky Street (Pollack, 1979). Before 1963, the presence of RFR was detected intermittently during routine surveillances of the building, and continuous monitoring of the signals was instituted in that year. A study of the health of U.S. personnel assigned to the Moscow embassy during the period from 1953 to 1976, compared with the health of those assigned to other U.S. Eastern European embassies, was conducted by Lilienfeld et al. (1978). The signal frequencies ranged from 2 to 7 GHz (Pollack, 1979), but the modulation patterns were not specified. The maximum incident average power densities and exposure durations varied with the period: 5 microwatts/cm^2 for 9 hours/day from 1953 to May 1975; 15 microwatts/cm^2 for 18 hours/day from June 1975 to February 7, 1976; and less than 1 microwatt/cm^2 for 18 hours/day thereafter. The highest average power density reported was 18 microwatts/cm^2 in one part of the southeast corner of the building, where beams from two sources converged.

After considerable effort spent in tracing employees and dependents, 1,827 employees and 1,228 dependents were identified as having been at the Moscow embassy during the 1953-1976 period. The control population consisted of 2,561 employees and 2,072 dependents assigned to embassies and consulates in Budapest, Leningrad, Prague, Warsaw, Belgrade, Bucharest, Sofia, and Zagreb during the same time period. Periodic tests for RFR at the control sites showed only background levels.

Medical records were reviewed for 1,209 Moscow employees and 834 dependents. The corresponding numbers for the control group were 1,882 and 1,507. Health questionnaires were returned by 969 Moscow employees and 1,129 control employees. The number of completed dependent questionnaires is not clearly specified in the report.

The authors of this study recognized and commented on the limitations placed on the study by their inability to acquire complete sets of medical records, death certificates, and returned health questionnaires, and by the imprecision of the classification of the individual employees according to probable extent of radiation exposure. Furthermore, they noted that the highest exposure levels were recorded late in the study and therefore, for the subgroup with the highest exposure, the period of time during which health effects might become apparent was the shortest. They also noted that the size of the study population was insufficient to detect excess risks that were less than twofold for many of the medical conditions studied. However, despite these acknowledged limitations, the authors were able to draw the following conclusions.

No discernible differences were found between the Moscow and control groups in total mortality or mortality from specific causes, nor were there differences in mortality between the Moscow and control groups of dependent children or adults. With the exception of cancer-related deaths among female employee groups (both Moscow and control), mortality rates for both Moscow and control groups were less than for the U.S. population at large. Although the study groups were subject to a large variety of health problems, the medical records indicate that these problems were shared nearly equally by both Moscow and control groups with two exceptions: the Moscow male employees had a threefold higher risk of acquiring protozoal infections, and both men and women of the Moscow group were found to have slightly higher frequencies of most of the common kinds of health conditions reported. However, the authors could not relate these two exceptions to RFR exposure. The health questionnaire information indicated higher incidences of some health problems in the Moscow employee groups than in controls: more correctable refractive eye problems, more psoriasis in men and anemia in women, and more frequent cases of depression, irritability, difficulty in concentrating, and memory loss. However, the authors note:

> In view of the possibilities which had been publicized of the increased danger to their health and that of their children, it is not at all surprising that the Moscow group might have had an increase in symptoms such as those reported. However, no relationship was found between the occurrence of these symptoms and exposure to microwaves; in fact, the four symptoms mentioned earlier, which showed the strongest differences between the Moscow and Comparison groups, were all found to have occurred most frequently in the group with the least exposure to microwaves. (Lilienfeld et al., 1978)

For dependents, the authors found no differences between the adult Moscow and control groups. The incidence of mumps in the Moscow dependent children was twice as great as that in the control children. The incidence of congenital anomalies in children born after arrival of

the parents at the duty station was comparable for the Moscow and control groups.

Finally, the authors summarized as follows:

> With very few exceptions, an exhaustive comparison of the health status of the State and non-State Department employees who had served in Moscow with those who had served in other Eastern European posts during the same period of time revealed no differences in health status as indicated by their mortality experience and a variety of morbidity measures. No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow embassy in the causation of any adverse health effects as of the time of this analysis. (Lilienfeld, et al., 1978)

Two studies have been made of the possible relationship between the occurrence of Down's syndrome (Mongolism) in Baltimore and presumed exposure of the fathers to RFR from radars during military service (Sigler et al., 1965; Cohen et al., 1977). The first study involved 216 Down's syndrome children and 216 control children matched for hospital of birth (or home birth), sex, date of birth, and maternal age at birth; the children were all born between January 1946 and October 1962. The data for this study were derived from Baltimore hospital records and interviews with the parents. These data showed that 63.1% of the case fathers and 56.6% of the control fathers had been in the military, but that 8.7% of the case fathers and only 3.3% of the control fathers had reported close association with radars (both within and outside of military service), a statistically significant difference. The authors concluded that "the only truly puzzling association is the suggested relationship between Mongolism and paternal radar exposure," and that "one can only speculate concerning possible mechanisms, but the association between Mongolism and radar exposure deserves further investigation."

In the second study (Cohen et al., 1977), the data from the first study, denoted as the "Original Series," were examined together with data regarding 128 additional matched pairs, denoted as the "Current Series." More detailed questions about RFR exposure and military service were incorporated in the Current Series questionnaires, and service record information on the fathers was acquired. An attempt was made to acquire similarly detailed data on the fathers of the Original Series. In addition, a chromosome study of the fathers was undertaken to determine whether there was any detectable residual damage in the chromosomes of the peripheral blood. After considering the more detailed exposure information, the following findings were reported for the Current Series: 15.7% of case fathers and 21.3% of control fathers had received radar exposure; combining the probably exposed with the definitely exposed groups, the corresponding values were 16.0% and

28.3%. The reevaluated Original Series values for definitely exposed fathers were 18.6% for case fathers and 15.2% for controls, and when probably exposed fathers were added, the values were 20.6% and 15.7%.

When the data from the Original Series and from the Current Series were combined, the values for case and control fathers were 17.4% and 17.5%, respectively, for definitely exposed, and 22.7% and 20.6% when "some" exposure was included. None of the foregoing comparisons showed statistically significant differences. The results of the chromosome studies have not been reported yet.

The authors concluded that the Current Series did not confirm the suggestions of the Original Series that the fathers of the Down's syndrome children had either an excess of radar exposure or a larger proportion of military experience. The authors note:

> In view of the suggestive findings of the original series with regard to a possible radar association. it was certainly necessary to investigate this question further. The initial steps were taken. A replication study was the simplest and least expensive immediate approach. Supplementing it with the independent search of service records added an objective approach eliminating any possible differential in parental responses. These methods have been attempted with inconclusive findings; it is now necessary to look to the prospective, longitudinal, surveillance studies to resolve the issue (Cohen et al., 1977).

In a study of personnel who had served in the Navy during the Korean War (Silverman, 1979; Robinette and Silverman, 1977), a group of approximately 20,000 persons was selected and classified as having had occupational exposure to RFR on the basis of their titles of Electronics Technician, Fire Control Technician, or Aircraft Electronics Technician; another group of about 21,000 persons was classified as not having had occupational exposure because of their titles of Radioman, Radarman, or Aircraft Technicians Mate. For brevity, the latter group was referred to as the control group, even though these personnel may have had some RFR exposure--presumably much less than the first group. Although comparison with an unexposed group would have strengthened the study, the two groups selected were presumably similar in terms of non-RFR factors. The study utilized only extant records, covering 1955 to 1976, of mortality and morbidity (both in service and later in Veterans Administration hospitals), and of both granted and disallowed requests for disability compensation.

The report by Robinette and Silverman (1977) provides only mortality results. which show 619 deaths from all causes for the occupationally exposed group versus 579 deaths for the control group; the difference is not statistically significant. The death rates for both groups were lower than those for the comparable age group in the

U.S. population at large. Examination of these decedent data in more detail showed a significantly higher death rate from trauma in the exposed group; however, many of the trauma-associated deaths resulted from military aircraft accidents, and a higher proportion of the exposed group had subsequently become fliers. The incidence of deaths associated with arteriosclerotic heart disease was significantly lower in the exposed group. No significant differences were noted between the two groups in terms of total mortality or in terms of mortality from any of about 20 assigned categories of causes of death.

Although the later report by Silverman (1979) does not furnish details regarding morbidity and other health-related aspects, she did state:

> Differential health risks associated with potential occupational exposure to radar in the Navy more than 20 years ago are not apparent with respect to long-term mortality patterns or hospitalized illness around the period of exposure, two endpoints for which there is virtually complete information for the total study group. Later hospitalization (in Veterans Administration facilities only) and awards for service-connected disability, the two other endpoints examined, provide incomplete information. While some significant differences among the occupational groups classified by level of potential exposure have been found with respect to all the endpoints studied, the differences could not be interpreted as a direct result of microwave exposure (Silverman, 1979).

Peacock et al. (1971) reported that an initial examination of birth certificates filed during the period from July 1969 to November 1970 from Dale and Coffee Counties, Alabama, in which Fort Rucker is located, indicated that the number of clubfoot cases among white babies was much larger than the expected statewide incidence. A more detailed study of this and other congenital anomalies in the six counties surrounding Fort Rucker (Calhoun, Henry, Butler, Jefferson, Dale, and Coffee) showed a higher rate of anomalies among babies born to military personnel than for the state as a whole. However, for nonwhite populations, only Calhoun County had a significant departure from the expected incidence. No interpretation in terms of causal factors for the excess incidences was given in the report.

Burdeshaw and Schaffer (1977) reanalyzed the Alabama birth record anomaly data for 1968-1972, but instead of using statewide averages as control data, they compared the Coffee and Dale County data with those of each of the other 64 Alabama counties on a score and rank basis. In addition, to acquire more detailed information on hospital characteristics and reporting procedures, they sent questionnaires to 46 Alabama hospitals. They used that information to predict expected values for Lyster General Hospital within Fort Rucker. They found that

the two highest hospital anomaly rates were from Fort Rucker and Maxwell AFB (both military aviation centers), and that 13 of 17 Alabama counties with anomaly rates in the upper quartile were in a contiguous band from southeast to west-northwest Alabama, which indicated the existence of a geographically distributed anomaly problem. However, they also found evidence against the conclusion that the anomaly incidence rate in the Fort Rucker area was unusually high: overall rates for Coffee and Dale Counties ranked only sixth and eighth among the 67 Alabama counties; at least 5 other Alabama hospitals reported anomaly incidences that were not significantly lower than those for Lyster Hospital; Lyster's overall rate was within predicted limits for hospitals with its characteristics; there was no clustering of residences of mothers with anomalous children in the vicinity of radar sites; carefully controlled surveys from other (non-Alabama) hospitals revealed anomaly incidences consistent with Lyster's; and significant time-clustering of anomalies at Lyster indicated a high reporting rate for one or two particular physicians. The authors concluded that the birth record data did not demonstrate that an unusually large number of infants with congenital anomalies were born to military personnel at Fort Rucker or to other residents in the immediate area.

Siekierzynski (1974) compared the health status and fitness for work of 507 persons in Poland occupationally exposed to pulsed RFR exceeding 200 microwatts/cm^2 average power density (other RFR characteristics not specified) with a group of 334 workers at the same installations exposed to less than 200 microwatts/cm^2. Clinical tests included ophthalmoscopic and neurologic examinations, supplemented by psychological tests and EEG recordings. No statistically significant differences between the two groups were found. In our opinion, the lack of more definitive RFR exposure data vitiates, but does not invalidate, the negative findings of this study; i.e., the results provide no evidence for RFR-induced effects on the health status of either group.

Kalyada et al. (1974) clinically examined a group of specialists in the USSR working with RFR generators in the 40-200 MHz range for 1 to 9 years and reported the occurrences of functional changes in the central nervous system described as vegetative dysfunction accompanied by neurasthenic symptoms. No organic lesions were found, but among the many specific changes reported were deviations in the physiochemical and functional properties of erythrocytes and leukocytes. The authors also conducted experiments with human volunteers and reported functional changes in the thermoregulatory and hemodynamic systems and in the thermal, optical, and auditory analyzers. However, no RFR intensity values were given for either the specialists or the volunteers; most of the findings were presented in narrative form, with no actual data; and the nature of the control group studied was not described. Consequently, this paper provides little basis for affirming or denying the occurrence of possible adverse effects of occupational exposure to RFR.

Klimkova-Deutschova (1974) surveyed various industrial worker populations in Czechoslovakia, including metal welders, steel factory workers, plastic welders, technicians operating radio or television transmitters, and people working in research institutes and other industries that involve exposure to RFR. Miscellaneous administrative staff members were studied for comparison. Frequencies varied according to the place of exposure, ranging from 0.5 to 150 MHz, 300 to 800 MHz, or 3 to 30 GHz. The power densities, where specified, ranged from 100 to 3,300 microwatts/cm^2. A sample of 352 workers was selected from 530 people considered. The findings included EEG anomalies (consisting of synchronized waves of high amplitude and slow rhythm) and biochemical changes (such as elevation of fasting blood glucose, serum beta-lipoprotein, and cholesterol). Changes in brain-wave patterns and in blood sugar, protein, and cholesterol levels were described as more pronounced in the people exposed in the 3 to 30 GHz range. Although the author states whether or not differences among groups for specific manifestations were statistically significant (at the 0.05 or 0.01 level), numerical results were not reported and statistical methods were not described.

Sadchikova (1974) presented clinical observations on the health status of two groups of USSR RFR workers. Those in the first group (1,000) were exposed to up to a few thousand microwatts/cm^2, whereas those in the second (180) were exposed to values rarely exceeding several tens of microwatts/cm^2, both at unspecified "microwave" frequencies. A group of 200 people of comparable backgrounds but presumably not exposed to RFR served as controls. Sixteen kinds of symptoms were reported, including fatigue, irritability, sleepiness, partial loss of memory, bradycardia, hypertension, hypotension, cardiac pain, and systolic murmur. In the higher-power-density group, the indices for 5 of the 16 symptoms were higher than those in the lower-power-density group; they were lower for 9 symptoms and about the same for the remaining 2. Incidences in the control group were lower than those in either exposed group for 15 of the 16 symptoms. A few subjects of the first group who worked under unspecified "unfavorable" conditions developed cataracts. Although bar graphs were included that show percentages of changes in the 16 symptoms among the 3 groups, statistical treatments of the data were not provided, so whether any of the reported differences were statistically significant cannot be ascertained. The occurrence of cataracts in the few who were working under "unfavorable conditions" must be interpreted as an indication of exposure to power densities in excess of the cataractogenesis threshold (see Section C.6.4).

Pazderova (1971) reported on the results of a battery of medical evaluations carried out on 58 employees of Czech television transmitter stations. Exposure frequencies were estimated to range from 48.5 to 230 MHz at field intensities equivalent to 0 to 22 microwatts/cm^2, with a mean exposure duration of 7.2 years (10.6 hours/workday). Electrocardiograms, heart and lung X-rays, erythrocyte sedimentation

rates, urinalyses, and liver function tests were conducted, as well as hematologic, serologic, ophthalmologic, neurologic, gynecologic, psychiatric, and psychological examinations. The only statistically significant finding was that the mean plasma protein levels were higher than "normal" values taken from the literature, a finding that the author describes as unexplainable. The appropriateness of the use of literature control values is highly questionable.

In a later study by Pazderova et al. (1974), the effects of RFR on blood protein levels were reexamined. In the 60 to 300 MHz range, 51 people were exposed to fields up to about 20 microwatts/cm^2; in the 3 to 30 MHz range, 19 people were exposed to about 1,000 microwatts/cm^2; and in the 640 to 1,500 kHz range, 39 people were exposed to about 800 microwatts/cm^2. A group of 59 workers served as controls, but the authors indicate that the only difference between exposed and control groups was that those included in the exposed groups had worked in irregular shifts, whereas more than half of the control group had worked only morning shifts. The results showed that the levels of blood proteins and their fractions were within normal physiologic limits, both the mean and individual values, but statistically significant differences were found between mean values for the exposed and control groups.

In our opinion, the absence in either study of a control group that had received virtually no RFR exposure renders questionable an interpretation that any differences found were due to RFR exposure. It is likely that the altered values of blood proteins (which were within normal limits) were caused by other factors.

Several retrospective epidemiological studies have been performed, notably by Zaret et al. (1961), Cleary et al. (1965), Cleary and Pasternack (1966), and Appleton (1973), to ascertain whether chronic exposure to RFR could cause cataracts.

Zaret et al. (1961) looked for eye defects in a group of 475 persons who were believed to have been exposed to RFR at 11 military and nonmilitary establishments; a group of 359 persons served as controls. The investigators found a slight but statistically significant difference in defect scores between the two groups, but they expressed some doubt regarding the full validity of the scoring method used.

Cleary et al. (1965) examined Veterans Administration Hospital records of 2,946 Army and Air Force veterans of World War II and the Korean War who had been treated for cataracts. A control sample of 2,164 veterans was selected. On the basis of military occupational specialties, they classified each individual as a radar worker, a nonradar worker, or one whose specialty could not be discerned. In the radar group, they found 19 individuals with cataracts and 2,625 individuals without cataracts; in the nonradar group, 21 individuals had cataracts and 1,935 did not. (The remaining 510 subjects were in the

unspecified occupational category.) These differences between the radar and nonradar groups are not statistically significant.

Cleary and Pasternack (1966) statistically analyzed the records of 736 microwave workers and 559 controls for minor lens changes, using a scoring range from 0 to 3. They reported that the defect scores increased with age for persons in both groups, but that the average score for the microwave group was significantly higher than that for the control group. They suggested that this finding is an indication that exposure to RFR may have an aging effect on the lens. However, no cataracts or decreases in visual acuity were found.

In the Appleton (1973) study, which covered a period of 5 years, military personnel identified as having been occupationally exposed to RFR from radar and communications systems were matched as closely as possible in age and sex with other military personnel on the same bases who had not been occupationally exposed. Several ophthalmologists independently examined exposed and control personnel (without knowledge of the group to which each individual belonged) for opacities, vacuoles, and posterior subcapsular iridescence, taken as diagnostic precursors of cataracts. Because of the complexity of the eye and the unavoidable judgmental aspects in the diagnosis of each examining ophthalmologist, each precursor was scored as either present or absent in each individual, and the binary data thus obtained were used for statistical analyses by age group and numbers of persons per age group. The results indicated that more people in older age groups exhibited these precursors, but the pooled data from several Army installations showed no statistically significant differences between exposed and control groups. The presence or absence of the three diagnostic precursors is only a crude measure of actual or possible incipient eye damage, useful primarily for statistical purposes.

Like those in other retrospective epidemiological studies, the accuracy and detail of the exposure histories (frequencies, intensities, durations, and so on) taken for either the exposed or the control groups in these three ocular studies is difficult to determine. However, it is quite likely that the exposed groups did receive more RFR exposure than the control groups.

In summary, none of the U.S., Polish, and Czechoslovakian epidemiologic studies analyzed here offers clear evidence of detrimental effects associated with exposure of the general population to RFR. However, the Soviet findings, which are consistent with the voluminous, earlier Soviet literature, suggest that occupational exposure to RFR at average power densities less than 10 mW/cm^2 does result in various symptoms, particularly those associated with CNS disorders. Because the USSR symptomatology has never been reported in Western studies and because of the marked differences between Soviet and Western publications in the procedures used for reporting data, any prediction

of possible RFR hazards based on the USSR epidemiological studies would require acceptance of these Soviet findings at face value. Thus, there is no reliable epidemiological evidence that chronic exposure of humans to the levels of RFR from OTH-B outside the exclusion fence will be hazardous to their health.

C.6.2 Mutagenesis and Cancer Induction

Over the past 30 years, several studies have been conducted on possible genetic and cytogenetic effects of RFR, and at least one published report has suggested the possibility of cancer induction by chronic exposure to RFR. Because cancer induction is considered to be related to mutagenesis (Ames, 1979), the two subjects are discussed together in this document.

Two studies of mutagenic effects of RFR in bacteria and yeasts gave negative results. The first study (Blackman et al., 1976) involved exposure of E. coli WWU to 1.70- or 2.45-GHz RFR at 2 to 50 mW/cm^2 for 3 to 4 hours. The second study (Dutta et al., 1979) involved exposure of S. Cervisiae D4 to 2.45-GHz RFR at 40 mW/cm^2 or to 8.5-9.6 GHz RFR at 1-45 mW/cm^2 for 120 min. Another part of the study involved exposure of S. typhimurium at the same frequencies and power densities for 90 min.

Four separate studies of mutagenic effects of RFR in fruit flies (D. melanogaster, a common mutagenic test animal) also gave negative results. The first study (Pay et al., 1972) involved exposure of 0 to 24-hour-old males to 2.45-GHz RFR at 6.5, 5.9, and 4.6 W/cm^2 for 45 min. The test consisted of mating the exposed males to females to determine effects of the RFR on fertility, and then remating the offspring to determine presence of recessive lethal mutations. The results were negative in both cases. The second study (Mickey et al., 1975) involved exposure of the flies to pulsed RFR at 20-35 MHz at an unstated power density level for 4 hours. The test consisted of observing for nondisjunction of X and Y chromosomes at mating, and the results again were negative. The third study (Dardalhon et al., 1977) involved exposure of the flies to 17 and 73 GHz RFR at 60 to 100 mW/cm^2 for 2 hours. No mutations were found. The fourth study (Hamnerius et al., 1979) involved exposure of fly embryos to 2.45-GHz RFR at 100 W/kg (200 mW/cm^2, approximately) for 6 hours. The test system consisted in measuring the frequency of somatic mutations for eye pigmentation. A positive control was included in the study (X-rays), and the authors concluded that the test system would have detected the mutagenic effect of 50 rad. No mutations were found in the RFR exposure.

Varma and coworkers conducted two studies on the induction of dominant lethal mutations in mice by RFR. The first study (Varma and Traboulay, 1976) involved exposure of the testes of Swiss mice to 1.7 GHz at 50 mW/cm^2 for 30 min or at 10 mW/cm^2 for 80 min. The second

study (Varma et al., 1976) involved a single exposure of the testes of Swiss mice to 2.45-GHz RFR at 100 mW/cm^2 for 10 min, 50 mW/cm^2 for three 10-minute exposures given in 1 day, or 50 mW/cm^2 for four 10-minute exposures given over 2 weeks. Because the studies were performed by the same principal investigator in the same laboratory and were reported at the same time, they are reviewed as a single study. The test consisted of breeding the exposed males to separate groups of unexposed females once each week for 7 to 8 weeks after irradiation. Females were killed on the 13th day of gestation and the uteri were scored for number of implants and number of resorption sites (dominant lethals). The authors concluded from the first study that the 1.7-GHz RFR was mutagenic under both conditions of exposure. They concluded from the second study that the 2.45-GHz RFR was mutagenic at the brief 100 mW/cm^2, but not at the 50 mW/cm^2 doses that were distributed over time.

These studies have a number of flaws. In the first study, errors were made in tabulating the data, leading to the question of how reliable the numbers presented may be. In the second study, the fetal late-mortality rates were significantly higher for the exposed animals than for the controls, raising the question of what other factors besides RFR might be causing dominant lethal effects. In both studies, systematic errors were made in the computation of the chi-square statistic used to evaluate the significance of the supposed mutagenic effect. If the chi-square is correctly computed, the first study shows a marginal, but significant increase in the number of lethal mutations for the study as a whole, but not for individual weeks of the study, and the second study shows no increase at all. In addition, the incidence of dominant lethal mutations in control animals differed significantly for the two studies (1% in the first, 5% in the second), leading to questions about the quality of the animal source and the reliability of scoring. If the controls from both studies are consolidated, no mutagenic effect can be demonstrated at any frequency, power density, or duration. Finally, the studies involved exposure of anesthetized animals, a condition under which the animals have no temperature control, and the temperature increase in the testes may have been much greater than what would be predicted from the exposure parameters.

Another study of dominant lethal mutations in rats (Berman and Carter, 1978) involved exposure to RFR at 425-MHz and 2.45-GHz RFR at power densities ranging from 5 to 28 mW/cm^2 for 3 hours daily (5 days/week) for periods of up to 3 months. The study found no evidence of impaired reproductive efficiency or increase in dominant lethal mutations.

Several studies on the induction of cytogenetic effects by exposure to RFR have been conducted. These studies usually involve two types of observations: (1) abnormalities in chromosomes (fragmentation, fusion, and interchromosomal bridges) at the metaphase stage of mitosis; and (2) sister chromatid exchanges.

The earliest study (Heller and Teixeira-Pinto, 1959) involved exposure of garlic root tips to 27 MHz for an unstated period of time. The text is brief and the description of exposure conditions is sketchy; from the description it can be calculated that the power density was somewhere between 2.5 and 600 mW/cm^2. Chromosome aberrations were reportedly found.

In a second study (Chen et al., 1974), Chinese hamster cells and human amnion cells were exposed in vitro to 2.45-GHz RFR at power density levels ranging from 200 to 500 mW/cm^2 for durations ranging from 1.5 to 20 min. A variety of chromosome aberrations were observed, but the incidence of aberrations did not increase with increasing power density level or duration of exposure, and the incidence in irradiated cells was not significantly different from that in control cells; hence one can conclude that RFR did not induce chromosome aberrations.

In a third study (Stodolnik-Baranska, 1974), human lymphocyte cultures were exposed to 2.95-GHz pulsed RFR (pulse characteristics not given) at 20 or 7 mW/cm^2 average power density for periods ranging from 10 min to 4 hr. Exposure to 20 mW/cm^2 for 10 min or longer reportedly produced chromosome aberrations, but no chromosome aberrations were reported from exposure to 7 mW/cm^2 for 4 hr. The author reported a "slight" temperature increase in cultures exposed to 20 mW/cm^2 but none in cultures exposed to 7 mW/cm^2. The results suggest that, if RFR does cause an increase in chromosome abnormalities, the effect may have a power-density threshold.

Mickey et al. (1975) exposed Chinese hamsters to pulsed K- and X-band RFR (approximately 18 and 10 GHz, respectively) at a power density level of between 200 and 500 mW/cm^2 for durations of up to 35 hr. Chromosome aberrations were reported in lung cells, bone marrow cells, and spermatogonia. The report does not describe the experimental protocol clearly, and reporting of control incidence of chromosome abnormalities is inadequate. Finally the calculated power density levels appear to be incorrect. It is unlikely that a Chinese hamster could survive the reported power density levels for longer than a few minutes.

Only one study of effects of RFR in sister chromatid exchange was found (Livinston et al., 1977). In this study, Chinese hamster ovary cells were exposed in vitro to 2.45-GHz RFR at unstated power density levels and durations. Sister chromatid exchanges were observed in RFR-exposed cells, but the same level of exchanges was produced in control cells by heating them to the same temperature as that produced by RFP exposure. The authors concluded that the production of sister chromatid exchanges is not related to RFR exposures.

Two published papers have implied an association between RFR exposure and cancer incidence. The first paper (Zaret, 1976) stated that the incidence of cardiovascular disease and cancer in the North

Karelian district of Finland had increased after completion of large radar stations immediately across the border in the USSR. The statement is not supported by any data or documentation; hence, from a scientific viewpoint it is of no value.

The second study (Prausnitz and Susskind, 1962) involved exposure of male mice to pulsed 9.27 GHz RFR at 100 mW/cm^2 average power density for 4.5 min/day, 5 days/week, for 59 weeks. Each day's exposure was equal to one-half of the acute LD_{50} of the animals. The study was well-designed and well-executed, and for its time can be considered an excellent model of a chronic toxicological study. The results of the study were as follows: (1) beginning at about 4 weeks into the study the mice showed increasing atrophy of the testes; (2) a number of them died during the exposure; (3) the death rate during exposure was greater in control than in irradiated animals--at the end of the exposure series, 50% of the control animals and 65% of the irradiated animals were still alive; (4) liver abcesses were found in some of the animals at necropsy, but because some of the tissues were lost by autolysis, the relative incidence in irradiated and control animals was indeterminate; (5) during irradiation a number of the animals developed leucosis, which was described in the paper as a "cancer of the white blood cells." The incidence of leucosis was greater in the irradiated than in the control animals.

The authors explained the testicular atrophy as resulting from chronic heating of the testes, an explanation that is highly reasonable. They attributed the deaths during exposure to a pneumonia infection accidentally introduced into the colony during the experiment. They suggested that the better survival of the irradiated animals was due to the protective effect of the daily rise in temperature ("fever") induced by the daily irradiation. The explanation is plausible, but not proven; RFR irradiation is known to have effects on the immune system. The liver abscesses do not merit specific comment. The greater incidence of leucosis in the animals during RFR irradiation appears real, but the interpretation appears faulty. Leucosis (also spelled "leukosis") is defined in the dictionaries of medicine and pathology as an abnormal rise in the number of circulating white blood cells. It is not defined as a form of cancer, though the dictionaries give detailed definitions of various types of leukemia, which <u>are</u> cancers of the circulatory system. Leucosis can arise from a number of causes, including stress, endocrine disturbances, and infection, such as that causing liver abscesses. In addition, two other factors must be considered: (1) In the irradiated animals, the incidence of leucosis was greater, but the survival of the irradiated animals was also greater. This would be considered unusual for most forms of mouse leukemia. (2) During irradiation, the incidence of leucosis was greater in the treated animals, but following irradiation, it was not. This would imply that a spontaneous remission of the "cancer" occurred after irradiation ceased. For true cancer, this would be considered quite improbable. Overall, the data do not provide any

evidence that chronic RFR exposure induces any form of cancer in the exposed animals.

Two other studies of chronic irradiation of animals have also been performed. The first study (Spalding et al., 1971) involved exposure of mice to 800-MHz RFR at 43 mW/cm^2 for 2 hr/day, 5 days/week, for 35 weeks. Some deaths occurred during the exposure, and were attributed to thermal effects caused by faulty positioning of the animal holders. The mean life span of the remaining irradiated mice was not different from that of the controls, general indications of health were the same in the two groups, and occurrence of cancer was the same in irradiated and control animals.

The second study (Baum et al., 1976) involved exposure of rats to electromagnetic pulses at a rate of 5/s continuously over a period of 94 weeks. The spectrum of the pulses corresponded to an RFR frequency of 450 MHz, and each pulse had an intensity of 447 kV/m. The exposures had no effect on blood chemistry, blood count, bone marrow cellularity, fertility, embryological development, cytology, histology, or occurrence of cancer.

In summary, there is no evidence that exposure to RFR induces mutations in bacteria or fruit flies. Belief that it induces mutations in mammals requires acceptance at face value of two studies that are demonstrably badly designed and executed and have numerous flaws in the reporting, analysis, and interpretation of the data. Evidence for cytogenetic effects of RFR is mixed. The lowest power density at which cytogenetic effects were reported was 20 mW/cm^2 (Stodolnik-Baranska, 1974), a value considerably higher than the levels from OTH-B outside the exclusion fence, but these results are contradicted by Chen et al. (1974), who failed to find cytogenetic effects at 200-500 mW/cm^2. There is no evidence that chronic exposure to RFR causes induction of any form of cancer, even at a power density of 100 mW/cm^2.

C.6.3 Studies on Teratogenesis and Developmental Abnormalities

In the narrowest sense of the word, teratogenesis refers to the production of anatomical aberrations in a developing fetus. The term is most often applied to the development of mammalian fetuses, but studies of development of eggs of birds and the pupae of the darkling beetle, Tenebrio molitor, have also been performed with RFR. Although the term usually refers to anatomical anomalies, studies have also included observation of fetal death and/or resorption and of physiological and cellular abnormalities in the offspring observed postpartum.

Two general remarks are pertinent to the various studies of teratogenesis produced by RFR. First, the mechanism by which terata are usually produced involves alteration (often temporary) in the rate of growth of a particular tissue under development. Development of the

entire fetus is a complex process requiring that individual tissues develop within a preset time frame, and interruption of this timing will result in abnormalities because a particular tissue or organ fails to complete development on schedule. Because of this, production of abnormalities is highly dependent on the time in the gestation sequence when the agent is applied and on the species of animal under study.

The second remark is that the experimental circumstances in studying development of birds' eggs or insect pupae differ from those for the study of mammalian teratogenesis. In the former instance, the experimental material is exposed to the whole environment without any protection; hence the studies must include rigorous control of all environmental parameters, including temperature. In the latter instance, the developing fetus is isolated from the environment to some extent by the dam; however, influences of the noxious agent on the dam must be considered as a potential indirect source of teratogenic effect.

Several studies have been conducted on RFR induction of teratogenesis in pupae of Tenebrio. Carpenter and Livstone (1971) exposed individual pupae to 10-GHz RFR in a waveguide at the equivalent of about 17 mW/cm^2 (SAR of 40 W/kg) for 2 hr or 68 mW/cm^2 for 20 or 30 min. As representative results, only 24% of the pupae exposed for 20 min developed into normal beetles; 25% died and 51% displayed gross abnormalities. About 90% of the sham-exposed pupae developed normally. Pupae were also radiantly heated to the temperature obtained with RFR, and about 75% emerged as normal beetles. The investigators therefore concluded that the abnormal development of RFR-exposed pupae cannot be explained as a thermal effect.

Lindauer et al. (1974), using pulsed and CW 9-GHz RFR at equivalent average power densities, also obtained statistically significant numbers of anomalies in Tenebrio exposed at 17.1 and 8.6 mW/cm^2. There were no significant differences in results for pulsed and CW RFR, and no clear dependence of the effect on dose rate or total dose was found.

Liu et al. (1975) extended this work at 9 GHz and found significant teratogenesis for 2-hr exposures at a power density as low as about 0.17 mW/cm^2. In addition, exposures at various levels and durations corresponding to a constant dosage of 4 mW-hr yielded evidence of reciprocity.

Green et al. (1979) found that pupae cultured and exposed at ambient relative humidities of less than 35% appeared to be more susceptible to RFR teratogenesis than pupae similarly treated at higher humidities. At the lower humidities, they observed a slight rise in the incidence of terata with increasing applied RFR power (2-hr constant exposure) up to 40 mW (34 mW/cm^2). At 320 mW, they observed a further increase in teratogenic frequency, accompanied by an increase in pupa death before completion of development. The investigators attributed the apparent "power window" at 80 mW to an antagonism between nonthermal

teratogenic effects and protective effects caused by the rise in temperature.

Pickard and Olsen (1979) used pupae from two sources. "Colony-pupae" were those derived initially as larvae from one supplier and raised on Purina dairy meal; "K-pupae" were purchased as larvae in three batches from another supplier and raised on Kellogg's Special K. Groups of K-pupae from the first batch and colony-pupae were sham-exposed or exposed at 6 GHz for 2 hr to either a standing-wave electric (E) field of 91 V/m (equivalent free-space power density of 2.2 mW/cm^2) or a magnetic (H) field of 1.53 A/m (88.3 mW/cm^2), or for 13 hr to a traveling-wave electromagnetic (far) field of 11 mW/cm^2. There were no significant differences in the frequencies of abnormalities between the groups exposed to the E field and the corresponding control groups of pupae of either type. However, the proportion of nonnormal beetles from the control K-pupae was significantly larger than that from the colony-pupae. In addition, exposure to the H field (of higher intensity) produced a significant effect on the K-pupae, but not on the colony-pupae. The H-field experiment was repeated with K-pupae from the other two batches, yielding RFR effects ranging from "doubtfully deleterious" to "significantly beneficial." Ambiguous results were also obtained from exposures for 13 hours at 6 GHz and 4 hours at 10 GHz. These variations appear to be due to uncontrolled differences in such non-RFR factors as the source of larvae, pupae maintenance regimes and handling protocols, the pupae containers used for pupation, and ambient temperature, an explanation that could account for the variabilities among the results of the other investigators cited above.

Pickard and Olsen (1979) nevertheless concluded that their reults indicate that RFR can be teratogenic in Tenebrio. However, the hypothesis of Carpenter and Livstone (1971) that the effect is nonthermal remains unproved. Specifically, Olsen and Hammer (1978) measured spatial distributions of absorbed RFR in pupae by thermographic imaging during irradiation at 1.3, 6, and 10 GHz, and found large local variations of SAR, which would not be obtained with the radiant heating used by Carpenter and Livstone.

Fisher et al. (1979) studied the development of chicken embryos in eggs exposed to 2.45-GHz RFR continuously for 4 or 5 days. The eggs were irradiated in 6 x 6 arrays. The power density ranged, over the array, from 1.4 to 6.2 mW/cm^2, with a mean of 3.46 mW/cm^2, and the exposures were done for incubator temperatures of 32 to 36 deg C. Control eggs were sham-exposed under similar conditions. Cranial lengths and wet masses of embryos were measured after exposure. At 36 deg C incubator temperature, cranial lengths and wet masses of RFR-exposed embryos were lower than those of the controls, but the rate of growth was greater for the former than for the latter. The converse was true for 32 deg C incubator temperature. The investigators observed no difference in incidences of sterility or premature death between the RFR and control groups. From the description of the method it is

difficult to determine whether the temperatures of the RFR-exposed eggs were actually measured or whether possible differences in temperatures of RFR-exposed eggs might have existed because of the spatial variation of power density. Finally, the significance of the finding in relation to possible human hazard is unclear because the embryos were not carried to hatching.

McRee and Hamrick (1977) exposed Japanese-quail eggs in 6 x 5 arrays to 2.45-GHz CW RFR at 5 mW/cm^2 (SAR about 4 W/kg) for 24 hr/day during the first 12 days of development. They found no gross deformities in the quail when euthanized and examined at 24-36 hr after hatching, and no significant differences in total body weight or the weights of the heart, liver, gizzard, adrenals, and pancreas between RFR- and sham-exposed groups. Hematological assays showed statistically significant higher hemoglobin and lower monocyte counts in the RFR-exposed birds, but no differences in the other blood parameters. The differences in mean temperature from egg to egg in the RFR-exposed arrays were as much as 0.5 deg C, rendering it difficult to associate these positive findings with RFR per se.

In another study (Hamrick et al., 1977), groups of eggs were similarly exposed and the birds were reared for 5 weeks after hatching. No significant differences in mortality or mean body weights at 4 and 5 weeks were found between RFR- and sham-exposed groups.

Teratogenic effects of RFR have been reported in a number of studies in rats and mice. The subject was reviewed recently by O'Connor (1980), who observed that, because of the high power density levels employed, the probability of killing the mother rat was somewhat larger than the probability of producing a teratogenic effect.

A major study in which a significant number of mouse terata were produced without significant mortality of the dams was that of Rugh et al. (1975, 1974). These researchers determined the average dose per unit mass (D/M) for lethality in female mice by exposing groups of CF-1 mice to 2.45-GHz RFR at 138 mW/cm^2 for various durations (at 23.5 deg C and 50% relative humidity). The mean D/M value was approximately 11 cal/g (10.65 for mice in estrus and 11.50 for mice in diestrus). They then exposed pregnant mice on day 8 of gestation at 123 mW/cm^2 for 2 to 5 min, corresponding to sublethal values of D/M ranging from 3 to 8 cal/g. On gestational day 18, the litters were examined for resorptions, and for dead, stunted, malformed, and apparently normal fetuses. A plot of the percentage of normal fetuses in each litter versus the value of D/M showed a considerable number of litters (too dense to count) with 100% normal fetuses (over the exposure range from 3.4 to 7.8 cal/g), 6 litters with no normal fetuses (over the range from 5.8 to 7.7 cal/g), and the remainder with various intermediate percentages. A similar plot of the percentage of resorptions per litter showed many with none (up to 7.7 cal/g), 3 with 100% (all above 6 cal/g), and the remainder with intermediate values. The incidence of

exencephaly (brain hernia) was also similarly plotted. Many litters showed none (spanning the entire dose range). The maximum was 60% (2 litters at about 7 cal/g), and the average expectancy at 8 cal/g was only 12%. Apparently no control mice were used, presumably under the assumption that the natural incidence of exencephaly is relatively rare.

For reasons that are not understood, Rugh et al. state that "...there seems to be no evidence of a threshold effect within the range of this study. It should be pointed out that data were not obtained below doses of about 2.5-3.0 cal/g, since preliminary studies at these lower radiation levels showed no teratogenesis." These two statements are contradictory; i.e., the second one implies the existence of a threshold. We used the data points of Figure 8 of Rugh et al. (1975) to determine the numbers of exencephalic fetuses in dose intervals of 0.5 cal/g over the range, assuming a mean of 10 fetuses per litter. Plotting the results showed the existence of a threshold of about 3.6 cal/g. A similar treatment of the points in Figure 7 of Rugh et al. (1975) showed a threshold for resorptions at about 3.5 cal/g. Although these values may be in error because of the large scatter of the data, the existence of thresholds is evident. However, in view of the small ranges of exposure duration and dose rate used, it is not clear that these thresholds are really for integrated doses; they may represent dose-rate thresholds instead.

Berman et al. (1978) exposed pregnant CD-1 mice in 5 x 5, 7 x 4, or 3 x 5 arrays to farfield 2.45-GHz RFR for 100 minutes daily on gestational days 1 through 17 at 3.4, 13.6, or 14.0 mW/cm^2, or on gestational days 6 through 15 at 28 mW/cm^2 (at 20.2 deg C and 50% relative humidity). Control mice were sham-exposed similarly. All mice were euthanized on day 18 and their uteri were examined for the number of resorbed and dead conceptuses and live fetuses. The live fetuses were examined for gross morphological alterations and weighed. Ten types of anomalies were tabulated by the numbers of litters affected. (The numbers of fetuses affected in each litter were not presented.) A total of 27 of the 318 RFR-exposed litters, irrespective of power density, had one or more live abnormal fetuses, versus 12 of the 336 sham-exposed litters. For most of the individual anomalies, the numbers of litters affected were either too small for statistical treatment or no RFR-related pattern was apparent. As an example of the latter, four litters exposed at 3.4 mW/cm^2 exhibited hematoma, with none in the corresponding sham-exposed group; however, two litters exposed at 13.6 mW/cm^2 and three sham-exposed litters were affected, and no litters were affected at 14.0 or 28.0 mW/cm^2, compared with one litter in each of their corresponding controls. By contrast, cranioschisis (akin to exencephaly or brain hernia) was exhibited by seven litters exposed to RFR--i.e., by one litter each at 3.4 and 13.6 mW/cm^2, three at 14.0 mW/cm^2, and two at 28.0 mW/cm^2--but by none of the control groups. However, there is no apparent pattern relating these numbers to power density. The investigators indicate that the number at each power density was not significantly different from zero, but that their sum

over all power densities (7 of 318 RFR-exposed litters versus 0 of 336 sham-exposed litters) was significant. The mean live fetal weights of the litters exposed at the three lower power densities were not significantly different from those of the corresponding sham-exposed litters; however, the mean weight of the litters treated at 28.0 mW/cm^2 was significantly lower than that of the sham-exposed litters.

These investigators used twin-well calorimetry to measure positional values of SAR in their arrays. For 5 x 5 arrays exposed at 10 mW/cm^2, the values of SAR ranged from 4.05 to 7.37 W/kg, possibly an indication of mutual RFR interactions among the mice. The authors correctly state that despite such variations, there was no overlap of SAR between arrays exposed at 13.6 and 3.4 mW/cm^2, corresponding to a power density ratio of 4:1. However, no SAR distribution data were given for the 3 x 5 arrays exposed at 28.0 mW/cm^2, or the 7 x 4 arrays exposed at 14.0 mW/cm^2, for which the power density ratio was only 2:1, thereby raising the question of possible SAR overlap in these experiments. Regarding abnormal fetuses, statistical treatment of the number of litters rather than the numbers of fetuses affected is of questionable validity. Also questionable is the summation of all litters exhibiting cranioschisis (irrespective of power density) and ascribing the statistically significant result to RFR exposure. Taken together, nevertheless, the results indicate that the levels of RFR used (which were not lethal to the dams) were marginally teratogenic to mice, a conclusion that is consistent with the findings of Rugh et al. (1975, 1974).

Chernovetz et al. (1975), in the first of two regimens, exposed one group of five pregnant C3H-HeJ mice to 2.45-GHz RFR for 10 min on day 11 of gestation, and one each of three other groups on days 12, 13, and 14 (totaling 20 dams). Each group was exposed concurrently in a multimode, mode-stirred microwave cavity (at about 22 deg C and 50% relative humidity), with the mice free to move about. At an estimated mean SAR of 38 mW/g, the energy absorbed was 22.8 J/g or 5.44 cal/g. The investigators stated that in a pilot study, 10-minute exposures at 40 mW/g (24 J/g or about 5.7 cal/g) were fatal to about 10% of a large number of pregnant mice, so that 38 mW/g was just sublethal. Another four groups were similarly sham-exposed. Eight other groups were injected with cortisone (a teratogen), four of which were similarly exposed to RFR and the other four were sham-exposed. All mice were euthanized on day 19, the numbers of implantations and resorptions were counted, and the fetuses were examined for structural abnormalities. There were no statistically significant differences in the percentage of fetal mortality or structural abnormalities between RFR and sham-exposed groups not administered cortisone, and no dependence on gestational day of treatment; however, the percentage of normal fetuses was 61% for those injected with cortisone and sham-exposed, and 50% for the cortisone-with-RFR groups. These percentages were significantly lower than those for the noncortisone group (both 81%), but they did not differ significantly from each other.

In the second regimen used by Chernovetz et al. (1975), which was designed primarily for a behavioral study, similar treatments were administered, but the exposures were done only on gestational day 14 and involved a total of 60 dams equally divided among the 4 treatments (RFR, sham-RFR, cortisone-with-RFR, cortisone-with-sham-RFR). All dams carried to term, and the numbers of pups that survived to weaning at postpartum age 21 days were noted. The results for the noninjected groups were 81 pups from those sham-exposed and 93 from those RFR-exposed, not a significant difference. From the cortisone-injected groups, the results were 25 pups from those RFR-exposed and only 2 from those sham-exposed. These values were significantly lower than those for the noninjected groups, and the difference between them was also significant. (The surviving pups were used in a behavioral study.)

These results indicate that absorption of about 5 cal/g of 2.45-GHz RFR is not teratogenic to mice, a finding that is at variance with those of Rugh et al. (1975, 1974) and Berman et al. (1978). Note also that the dosage for lethality reported by Chernovetz et al. was about 5.7 cal/g, or about half the mean value found by Rugh et al., hence their conflicting characterizations of doses with respect to lethality. Among the possible reasons for these apparently contradictory findings are the respective differences in exposure systems (cavity versus waveguide), the use of multiple versus individual animal exposures, gross uncertainties in actual doses, the mouse-strain difference (C3H/HeJ versus CF-1), variations in dam handling, and differences in gestational day of treatment (day 11 through 14 versus day 8). Also, Chernovetz et al. found fetal anomalies in about 20% of their control mice, whereas Rugh et al. apparently used no controls. Both groups of investigators indicate that extrapolation of their findings to higher mammalian species is an open question subject to experimental validation, a statement with which we concur.

In the first of two studies, Stavinoha et al. (1976, 1975) exposed 4-day-old mice in plastic containers for 20 min at 10.5, 19.27, or 26.6 MHz in a coaxial rectangular waveguide system, in which the electric field was 5.8 kV/m. Control groups were kept in similar containers outside the exposure chamber. The mice were weighed daily for the next 21 days. Graphs of weight versus age for the three frequencies showed virtually no differences between exposed and control animals. In the second study, litters of 4-day-old pups from 20 female mice were divided into three groups: (1) control pups; (2) thermal-control pups, held at 37 deg C for 40 min/day on 5 consecutive days; and (3) irradiated pups, exposed at 19 MHz for 40 min/day on 5 consecutive days in a near-field synthesizer, in which the electric field was 8 kV/m, the magnetic field was 55 A/m, and the two fields were in coincident planes. The pups were weighed daily before each treatment and until they were 21 days old, at which time the males and females were separated. Subsequently, the mice were weighed weekly for a total of 16 weeks. Statistical analyses of growth curves showed no significant differences among the three groups either for the males or

the females. As the investigators pointed out, although the fields used were very intense, relatively little RFR energy was absorbed by the mice because their sizes were very much smaller than the wavelengths used. Thus, it is inappropriate to apply these negative findings to humans exposed at frequencies in the same range.

Dietzel (1975) exposed pregnant rats abdominally at 27.12 MHz with a diathermy machine and applicator to 55, 70, or 100 W for up to 10 min on 1 day between gestational days 1 and 15. The rectal temperature of each rat was monitored during exposure, and the rat was removed from the field when its temperature reached 39, 40.5, or 42 deg C (in lieu of any other dosimetry). On day 20, the fetuses were removed, counted, weighed, and examined for malformations. Typical predominant abnormalities included neurocranial malformations from RFR exposure on days 9 and 10, kinked or short tails and "hand" defects for days 13 and 14, and cleft palate for day 15. The maximal numbers of abnormalities occurred for exposure on days 13 and 14, and correlated well with rectal temperature, indicating that the abnormalities resulted from heating by the RFR. The lack of adequate dosimetry renders it difficult to compare these results with those of other investigators. In addition, because of the high intensities of RFR used, the relevance of these findings to possible teratogenesis in humans exposed to much lower levels of RFR in the HF range is questionable.

Chernovetz et al. (1977) exposed pregnant rats for 20 min on only 1 day during gestational days 10 through 17 to 2.45-GHz RFR in a multimode, mode-stirred microwave cavity at a mean SAR of 31 W/kg in an ambient temperature of 22 deg C and 50% relative humidity. They also exposed rats to infrared radiation (IR) in an incubator at 47 deg C and 10-15% relative humidity. The incubator ambient was selected to produce the same colonic temperature rise of 3.5 deg C as the RFR exposure. Control groups were sham-irradiated in the microwave cavity.

A total of 64 pregnant rats was studied. Three dams died after IR exposure, seven died after RFR exposure, and none died in the control group. On day 19 of gestation, the 54 surviving dams were euthanized and the numbers of implantations and resorptions were counted. In addition, each fetus was examined for morphological abnormalities and its viability and mass were determined. The percentages of living fetuses per dam were about 98% each for the control and IR groups and 87% for the RFR group; this is a statistically significant decrease. The mean fetal mass for the control groups was 1.63 g, and the values for the IR and RFR groups were 1.53 and 1.54 g, respectively, both significantly lower than the mean for the control groups. No structural abnormalities were evident in any of the 468 formed fetuses, all of which were alive when taken, but severe edema and hemorrhagic signs were endemic in the IR and RFR groups. The brains of 60 fetuses were assayed for norepinephrine (NE) and dopamine (DA) in four groups each of five pooled brains from control, IR-exposed, and RFR-exposed animals. The average level of NE for the RFR group was significantly lower than that

for the controls, but ony marginally lower than that for the IR group. The averaged levels of DA ranked similarly, but the differences were not statistically significant. In their discussion, the authors concluded that "considered in sum, our findings could be taken as evidence that a brief but highly thermalizing application of 2,450-MHz microwaves or of infrared energy have biological effects both comparable and different when averaged colonic temperature changes are equal."

One of the problems with this investigation was the small number of rats involved (a point recognized by the investigators), which necessitated averaging the data in each group over the 10-16 day gestational period, a questionable procedure both biologically and statistically. Perhaps a minor point was the use of the sham-exposed rats as controls for the IR group instead of a separate set of sham-IR controls, in view of the relative humidity difference. Because of such problems, it is difficult to assess the validity of either the positive or negative results of this investigation.

In a study designed primarily for seeking possible effects of chronic RFR exposure on mother-offspring behavioral patterns and the EEG, Kaplan et al. (1980) exposed 33 female squirrel monkeys near the beginning of the second trimester of pregnancy to 2.45-GHz RFR in multimode, mode-stirred microwave cavities at whole-body SARs of 0.034, 0.34, or 3.4 W/kg (the last value equivalent to about 10 mW/cm^2 of plane-wave RFR) for 3 hr/day, 5 days/week until parturition. Eight pregnant monkeys were sham-exposed for the same periods. After parturition, 18 of the RFR-exposed dams and their offspring were exposed to RFR for an additional 6 months, and then the offspring were exposed without the dams for another 6 months. No differences were found between RFR- and sham-exposed dams in the numbers of live births or in the growth rates of the offspring. The major difference between RFR- and sham-exposed offspring was that four of the five exposed at 3.4 W/kg both prenatally and after birth unexpectedly died before 6 months of age. Although the numbers of animals used in the behavioral and EEG studies were adequate, the mortality values were too small to place much confidence in statistical inferences. Moreover, a follow-up study of mortality per se (Kaplan, 1981), which involved sufficient numbers of squirrel monkeys for adequate statistical treatment, did not confirm the RFR-induced offspring mortality results.

In summary, the investigations of RFR-induced teratogenesis and developmental abnormalities support the conclusion that such effects result from the heat produced by the RFR rather than from any special teratogenic properties of RFR. Thus, there is no evidence that the RFR from OTH-B beyond the exclusion fence is potentially hazardous to children in utero at any stage of development.

C.6.4 Ocular Effects

The fear that RFR can cause cataracts is a recurring theme in newspapers and other popular media. Indeed, based on many investigations with animals by various researchers during the past 30 years, it is undoubtedly true that if a person's eyes were exposed to intensities high enough to elevate the temperature of the lens of the eye by about 5 deg C (9 deg F) or more, the lens would quickly suffer damage. The lens is the region of the eye most vulnerable to RFR because other regions have more effective means of heat removal, such as greater blood circulation, evidenced by much smaller temperature elevations in these regions than in the lens at the same incident power density. Therefore, the basic controversy regarding ocular effects is centered on whether exposure to much lower intensities (i.e., to power-density levels that would produce much smaller lens-temperature elevations) for long periods of time, either continuously or intermittently, can cause eye damage. Implicit in this controversy is the issue of whether effects (if any) of long-term, low-level exposure in the eye are cumulative, as are the effects of continual or repeated ingestion of certain toxic substances in individual doses that are well below rapid-toxicity levels.

C.6.4.1 Humans

Some cases of ocular damage in humans ascribed to occupational exposure to RFR were reported during the 25 years after World War II (Zaret, 1969; Shimkovich and Shilyaev, 1959; Hirsh and Parker, 1952). Although the exposure histories of these individuals could not be ascertained with any degree of certitude, it is likely that their actual or incipent vision impairment resulted from exposure to average power densities substantially greater than the threshold found in animal studies (about 150 mW/cm^2).

More recently, the occurrence of cataracts in two editors with the New York Times was ascribed, in newspaper accounts during 1977 and 1978, to their exposure to supposed RFR from the cathode-ray tubes in video-display terminals used by them (Justesen, 1979). Cases of RFR-induced birth defects and abortions were also linked, in other newspaper stories, to exposure to video terminals. The New York Times arranged for measurement surveys of the terminals in question. These surveys yielded negative results; the only measurable radiations emitted by the terminals were well above the RFR spectrum. Independent surveys of the same terminals by personnel from NIOSH (1977, cited in Justesen, 1979) confirmed these findings.

Retrospective epidemiological studies have been conducted, notably by Zaret et al. (1961), Cleary et al. (1965), Cleary and Pasternack (1966), and Appleton (1973), to determine whether prolonged exposure to RFR is cataractogenic. These studies are discussed in Section C.6.1.

C.6.4.2 Animals

During the past 30 years, various investigations have been conducted on the effects of RFR exposure on the eyes of live experimental animals. Many of the results indicate that intraocular temperature increases of about 5 deg C or more are necessary for eye damage (Guy et al., 1974; Williams et al., 1955; Daily et al., 1952; Richardson et al., 1948). Also lens opacifications caused by RFR exposure alone were not produced at the same average power density when the eye was cooled during exposure (Kramar et al., 1975; Baillie, 1970; Baillie et al., 1970).

Many of the results of RFR exposure indicate the reciprocity (inverse relationship) between average power density and exposure duration for cataract formation and the existence of a threshold average power density of about 150 mW/cm^2 for single or multiple exposures for tens of minutes or more (Carpenter, 1977; Ferri and Foti, 1977; Guy et al., 1974; Williams et al., 1955). As a representative example (Guy et al., 1974), for average power densities decreasing from about 500 to 200 mW/cm^2, the exposure duration needed to cause eye damage in the rabbit increased from 1-2 minutes to about 20 minutes. Also, cataracts were not produced at 100 mW/cm^2 for exposure durations of up to at least 100 minutes (exposures for longer periods were not done in this investigation). Thus, curves of average power density versus exposure duration show that the average power density for cataractogenesis asymptotically approaches a value of about 150 mW/cm^2. Cataracto-genesis thresholds of comparable magnitude are evident from the experimental results of others (Carpenter, 1977; Birenbaum et al., 1969; Williams et al., 1955). Carpenter (1979), in reviewing RFR cataractogenesis from a clinical viewpoint, presents a detailed description of the post-exposure progression of eye changes, based on experimental results with animals.

Kramar et al. (1975) measured intraocular temperatures *in vivo* at 200 mW/cm^2 over a period of 40 minutes by quickly inserting a thermocouple probe during brief shut-off of the RFR at the end of successive 5-minute exposure periods. They found that the temperature of the vitreous humor rose from about 37 deg C to about 42 deg C during the first 10 minutes of exposure and remained at 42 deg C for the rest of the exposure period. Equilibrium between the rates of energy absorption and heat removal in that region of the eye is believed to be the determining factor in attaining the 42 deg C plateau temperature. In the orbit, which is cooled by blood flow to a greater extent than the vitreous humor, the corresponding plateau temperature was less than 40 deg C.

The effects of temperature increases produced by non-RFR means on isolated (*in vitro*) rat lenses were studied recently by Stewart-DeHaan et al. (1979). Lenses incubated in tissue culture medium M 199 (containing 10% fetal calf serum) at the normal physiological

temperature of 37 deg C maintained their clarity for up to 2 weeks. When 10 times the normal concentration of serum glucose was included in the medium, opacities and associated globular degeneration of the lens cells developed in 1 day. Similar effects were observed when lenses were warmed to 39 or 41 deg C for 1 hour and then incubated at 37 deg C for 24 hours. Lenses heated to 60 or 75 deg C for 1 hour did not become opaque. The authors surmise that the lenses became histologically "fixed." Radioactive tracer studies by these investigators suggest that membrane changes may be involved in temperature-induced cataract formation.

A number of investigators (Ferri and Foti, 1977: Reider et al., 1971; Richardson et al., 1951) compared the ocular effects of pulsed and CW RFR at equivalent average power densities. In representative investigations, the average power densities were greater than 100 mW/cm^2 and the exposures were for about 1 hour/day for several weeks. No significant differences between the effects of pulsed and CW RFR were found.

The existence of a cataractogenesis threshold of about 150 mW/cm^2 is regarded as evidence that single or multiple exposure for indefinitely long durations at average power densities well below the threshold would not cause eye damage to humans or any other species. This conclusion is supported by preliminary results of an investigation by Chou et al. (1978). They exposed one group of six rabbits to 2.45-GHz CW at 1.5 W/cm^2 for 2 hr/day over several months, and another group to pulsed RFR at the same frequency and average power density (10-microsecond pulses at a repetition rate of 100 pulses/s, comprising a duty cycle of 0.001 and a pulse power density of 1.5 W/cm^2); a third group was sham-irradiated. Periodic eye examinations for cataract formation yielded no statistically significant differences among the three groups.

These results from animal studies indicate that RFR cataractogenesis is essentially a gross thermal effect that has a threshold power density at which the difference between the rates of heat generation by RFR and heat removal is large enough to result in damage to the lens of the eye. The mean threshold values probably vary to some extent from species to species, but are of the order of 100 mW/cm^2. Thus, chronic exposure to the RFR from OTH-B beyond the exclusion fence is most unlikely to result in eye damage because the power densities there are far below the threshold.

C.6.5 Studies of the Nervous System

Several types of studies have been conducted on effects of RFR on the nervous system of animals. These studies are considered particularly important in the USSR, where RFR is believed to stimulate the nervous system directly and thereby cause a variety of physiological effects. Scientists in the United States tend to doubt that RFR

interacts directly with the nervous system except, possibly, under special circumstances (to be discussed later in this section), and they consider most effects of RFR on the nervous system to be indirect results of other physiological interactions.

C.6.5.1 RFR Hearing Effect

Humans in the vicinity of some types of pulsed radar systems have perceived individual pulses of RFR as audible clicks (without the use of any electronic receptors). This phenomenon, first investigated by Frey (1961), has attracted much interest--especially in the United States--because it has often been cited as evidence that nonthermal effects can occur and because an initial hypothesis was that one of the possible mechanisms for perception is direct stimulation of the central nervous system by RFR. Various theoretical and experimental studies, the latter with both human volunteers and laboratory animals, have been conducted to determine the conditions under which pulsed RFR is audible and to investigate the interaction mechanisms involved. Many of the results support the hypothesis that a pulse of RFR having the requisite pulse power density and duration can produce a transient thermal gradient large enough to generate an elastic shock wave at some boundary between regions of dissimilar dielectric properties in the head, and that this shock wave is transmitted to the middle ear, where it is perceived as a click. Persons with impaired hearing are unable to hear such clicks, and experimental animals in which the cochlea (the inner ear) has been destroyed do not exhibit brainstem-evoked responses. Therefore, it is unlikely that this phenomenon occurs because of direct RFR stimulation of the brain. Also, because individual pulses can be perceived, citations of low average power densities based on calculations for two or more widely spaced pulses are not particularly meaningful. Representative investigations of this phenomenon are summarized below.

White (1963) reported that acoustic transients can be generated in various metals and plastics, in a piezoelectric crystal, and in liquids such as water by transient surface-heating with pulses of RFR (or from an electron beam). Such transients were detectable in a barium titanate crystal at calculated surface temperature rises of the order of only 0.001 deg C.

Foster and Finch (1974), using 2.45-GHz RFR, confirmed White's findings that such transients can be produced in water, and they measured the audiofrequency pressures generated by several combinations of pulse power density and pulse duration. The results indicated that the peak pressures would be sufficient to induce human perception of such RF pulses as auditory clicks. They also showed that the effect vanished when the water was cooled to 4 deg C, at which its thermal expansion coefficient is very small. These results support the thermoelastic hypothesis.

Lin (1977a, 1977b) has reported on detailed theoretical studies of the RFR auditory effect. He assumed that the auditory sensation results from acoustic waves generated in the tissues of the head by rapid thermal expansion of the tissues upon microwave absorption, in consonance with the investigations cited above. His results indicate that the audio frequencies produced are independent of the frequency of the RFR, but are dependent on head size. The predicted fundamental frequency is 13 kHz for an adult human and 18 kHz for an infant.

Chou et al. (1977) studied cochlear microphonics (CM) produced by pulsed 918- and 2,450-MHz RFR in guinea pigs and cats. They found that the CM frequencies correlated well with the longest dimension of the brain cavities of the two species, but poorly with other brain cavity dimensions. Extrapolation of the results indicates that CM frequencies in humans should be between 7 and 10 kHz, in reasonable agreement with Lin's theoretical calculations.

Chou and Guy (1979b) used 918-MHz RFR to investigate the thresholds for brainstem-evoked responses in guinea pigs. They found that for pulsed durations of 10, 20, and 30 microseconds, the threshold incident energy density was approximately constant (1.56-1.87 microjoules/cm^2 per pulse), corresponding to incident power densities of 156, 78, and 62.4 mW/cm^2, respectively. However, for pulse durations longer than 70 microseconds, the incident pulse power density necessary for obtaining responses was approximately constant (about 90 mW/cm^2), representing corresponding increases of incident energy density per pulse with pulse duration. Chou and Guy indicated that their experimental results agree well with the predictions of the thermal expansion theory.

Chou and Galambos (1977) investigated the effects of external-ear blocking, middle-ear damping, and middle-ear destruction on brainstem-evoked responses to both acoustic and RFR stimuli. They found that only animals with intact cochleas were capable of responding to pulsed RFR.

Lebovitz and Seaman (1977) reported on single auditory unit responses to pulsed 915-MHz RFR and acoustic clicks. They found that the response of a typical single auditory unit was very similar to the response of the unit to acoustic click stimuli, differing primarily only in amplitude.

Cain and Rissman (1976, 1978) used 3.0-GHz RFR to study the auditory effect in two cats, two chinchillas, one beagle, and eight human volunteers. For the animals, surface or brainstem-implanted electrodes were used to measure the responses to RFR pulses and the responses evoked by audio clicks from a speaker. They found that the threshold energy density for RFR responses ranged from 8.7 to 14 microjoules/cm^2 per pulse for the cats, from 7.5 to 20 microjoules/cm^2 for the chinchillas, and that it averaged 5.0

microjoules/cm^2 for the beagle. For a pulse width of 10 microseconds, these values correspond to pulse power densities of 1.3 W/cm^2 for both cats, 1 and 2 W/cm^2 for the two chinchillas, and 300 mW/cm^2 for the beagle. The eight humans were given standard audiograms. Because such audiograms do not test hearing above 8 kHz, binaural hearing thresholds were also determined for seven of the subjects for frequencies in the range from 1 to 20 kHz. Five of the subjects could detect 15-microsecond pulses as clicks; the other three required a pulse duration of 200 microseconds for perception. No correlation between the results and the audiograms was apparent; however, there was a strong correlation between RFR perception and hearing ability above 8 kHz as determined from the binaural thresholds. The average threshold energy density for the humans was 10.6 microjoules/cm^2 per pulse. For 15-microsecond pulses, this value corresponds to a pulse power density of about 700 mW/cm^2; however, three of the subjects were able to perceive 15-microsecond pulses at a pulse power density of 300 mW/cm^2.

Tyazhelov et al. (1979) reported some peculiarities regarding auditory perception of pulsed 800-MHz RFR in humans. The pulse widths used ranged from 5 to 150 microseconds at pulse repetition frequencies (prfs) of 50 to 2,000 (the latter for short pulse durations). Each subject could be presented with sinusoidal audiofrequency (AF) sound waves alternately or concurrently with the pulsed RFR and could adjust the amplitude, frequency, and phase of the AF signal to match the timbre and loudness of the perceived RFR. Using AF signals, the high frequency auditory limit (HFAL) of each subject was tested first. Three of the subjects who had HFALs above 10 kHz could not perceive such pulses. These results are consonant with those of Cain and Rissman. Among the peculiarities noted by Tyazhelov et al. was the biphasic dependence of RFR perception thresholds on pulse width. They also reported that subjects could detect beat frequencies when concurrently presented with 8-kHz AF sound and RFR having prfs above or below 8 kHz, and that when the prf was the same as the AF, the subject could cancel perception by adjusting the phase of the AF. Their conclusion is that the thermoelastic model is inadequate to explain these observations.

Frey and Coren (1979), using dynamic time-averaged interferometric holography, endeavored to detect tissue movement in successive layers of heads of animals exposed to pulsed RFR; for comparison, they used holograms obtained for the same animals during sham exposure. No movements were detected. The authors concluded that perception of pulsed RFR is most likely due to thermoelastic expansion within the cochlea rather than anywhere else in the head. However, the adequacy of the sensitivity of this holographic technique for detecting such movements is in dispute (Chou et al., 1980; Frey and Coren, 1980).

The occurrence of the RFR-hearing effect is well established, but is of no concern with regard to OTH-B because the RFR therefrom is not pulsed.

C.6.5.2 Calcium Efflux

Over the last 6 years, Adey and Bawin and their colleagues have reported extensively on their studies of changes in radioactive-calcium-ion ($^{45}Ca^{++}$) efflux from neonate chick brain preparations and isolated samples of cat cortex under very specific frequency and power-density regimes of unmodulated sub-ELF fields and of amplitude-modulated VHF and UHF fields. Recent reviews of this work are given in Adey (1980, 1979), and details of the experimental protocol are given in Bawin and Adey (1976, 1977). Briefly, following decapitation, forebrain hemispheres of neonate chicks were obtained by rapid dissection. The hemispheres were separated and one was used for exposure and the other as control. Each was incubated in a specified physiological solution containing $^{45}Ca^{++}$ for 30 min. At the end of incubation, the samples were rinsed three times with nonradioactive solution, transferred to new glass test tubes, bathed in 1.0 ml of solution, and exposed or sham-exposed for 20 min. Sets of 10 brain samples (10 exposed hemispheres, 10 control hemispheres) were used simultaneously. At the conclusion of exposure, aliquots of 0.2 ml of the bathing solution were taken, and their radioactivity was assayed by scintillation counting. Radioactivities (counts per minute, cpm, per gram) were normalized to the mean value of counts obtained in control effluxes. All normalized data were compared (by t-test) with matched samples of control values.

Adey (1977, 1978) presents data from experiments with approximately 190 chick brains for 450-MHz exposures. Power densities of 50, 100, 500, 1,000, 2,000, and 5,000 microwatts/cm^2 were used for 16-Hz amplitude modulation of the field. Statistically significant increases in $^{45}Ca^{++}$ efflux were seen at 100, 500, and 1,000 microwatts/cm^2, but not at 50, 2,000, or 5,000 microwatts/cm^2.

In a subsequent paper, Bawin et al. (1978) described experiments aimed at a better definition of the calcium sites responding to weak electrical stimulation. Changes in calcium efflux were studied with and without imposed RFR (450 MHz, 16-Hz amplitude modulation, 375 or 2,000 microwatts/cm2) to ascertain the effect of calcium concentration in the exchanging medium. Also tested were the effects of changes of pH, and the use of bicarbonate-free solutions. They also examined modification of calcium release, by the addition of lanthanum to the bathing solution, for both no-field and with-field stimulation conditions. Efflux of $^{45}Ca^{++}$ in the standard physiological solution was the "control" for these experiments.

The results confirmed the previous findings by Bawin and Adey that amplitude-modulated 450-MHz fields can stimulate the release of preincubated $^{45}Ca^{++}$ from isolated brain tissue. All of the results are stated by the authors to support the hypothesis that a limited number of extracellular cationic binding sites are involved in the transaction of weak extracellular electrical events.

Bawin et al. (1977) have also reported on results from a preliminary study involving the monitoring of calcium efflux from the intact cortex of 12 awake, paralyzed cats. The methods were similar to those used in the chick brain experiments. The cats were exposed for 20 min to 450-MHz fields amplitude-modulated at 16 Hz. Power densities were 375 or 1,000 microwatts/cm^2. Results are stated to be a clear increase in $^{45}Ca^{++}$ efflux during and following the exposure in 8 of the 12 animals. However, some animals apparently responded to the presence of the experimenter during sampling. Further experiments are stated to be in progress to remove the possibility of artifact and to elaborate on these preliminary findings.

Blackman et al. (1979) conducted experiments that verified and extended Bawin and Adey's findings for chick brain at 147 MHz. Treated tissue was exposed in a Crawford chamber to power densities between 500 and 2,000 microwatts/cm^2 and amplitude modulation of the carrier at selected frequencies between 3 and 30 Hz. They found a statistically significant increase in calcium efflux when the frequency modulation was 16 Hz and the power density was 750 microwatts/cm^2. (Preliminary findings indicating the existence of the power window, in addition to the frequency window discovered by Adey and Bawin, were reported by Blackman et al., 1977). The 750 microwatts/cm^2 value was corrected to 830 microwatts/cm^2 in Blackman et al. (1980).

Blackman et al. (1980) extended this work for 147-MHz RFR, amplitude-modulated (greater than 95%) at 9 Hz or 16 Hz, to examine a potential artifact introduced by the number of samples simultaneously exposed. Brain tissues were from 1- to 7-day-old chicks. Tissues were exposed for 20 min with 0-, 9-, or 16-Hz sinusoidal modulation at power densities of 110, 550, 830, 1,100, 1,380, and 1,660 microwatts/cm^2. One-half of each chick brain was exposed to the RFR, and the other half, which was neither exposed nor sham-exposed, served as the control. Halves of other brain pairs were sham-exposed, and the corresponding halves served as controls. In one series, four brain pairs were treated at the same time. In another series, four brain pairs and six dummy loads were treated simultaneously. Statistically significant differences of normalized calcium efflux were found between exposed and sham-exposed tissues for eight of the combinations of power density, modulation frequency, and number of tubes (brains plus dummy loads). These combinations were: 830 microwatts/cm^2 for 16 Hz and 4 tubes/chamber; 550, 830, 1,110, and 1,380 microwatts/cm^2 for 16 Hz and 10 tubes/chamber, and 550, 830, and 1,110 microwatts/cm^2 for 9 Hz and 10 tubes/chamber. For unmodulated exposure at 830 microwatts/cm^2, no differences were found between the efflux values for exposed- and sham-exposed groups. Two aspects of the results are puzzling: sham-exposed brain halves inside the transmission line generally gave higher values for calcium efflux than their corresponding control halves outside the transmission line, and the four-tissue-plus-six-dummy-load configuration gave a broader power-density window than the four-tissue-

without-dummy-load configuration. The reasons for these discrepancies are unresolved.

To determine whether changes in carrier frequency altered the range of power densities effective in producing statistically significant alterations in calcium efflux, Blackman et al. (1981) conducted experiments at 50 MHz, amplitude-modulated at 16 Hz. Exposure conditions and protocol were similar to those previously used by these investigators. The results of a power-density series demonstrated three effective power densities: 1,440, 1,670, and 3,640 microwatts/cm^2. No statistically significant effects were found at 720, 2,170, and 4,320 microwatts/cm^2. Of interest was the calculation that peaks of positive findings were associated with nearly identical rates of energy absorption, 1.4 microwatts/g at 147 MHz and 1.3 microwatts/g at 50 MHz.

Joines and Blackman (1981) modeled the 16-Hz amplitude-modulated exposure conditions of Adey at 450 MHz and of Blackman at 147 MHz and 50 MHz. Calculations showed that the average electric-field intensity within a spherically modeled sample remained the same at different carrier frequencies if the incident power density was adjusted by an amount that compensates for the change in complex permittivity of the brain tissue and for the change of internal wavelength as a function of carrier frequency. When this was done, all positive and negative results obtained at these three frequencies, when compared by average electric-field intensity within the sample, were in agreement. No result, positive or negative, was contradicted by a corresponding experimental result at a different carrier frequency. However, the model did not take into account the amplitude-modulation frequencies per se. Because not all modulation frequencies are effective, comparisons among average electric field intensities within the samples cannot be extended to other modulation frequencies. The mechanisms whereby modulation effects are mediated are speculative.

The foregoing studies were all concerned with nervous system tissue. In two studies, the calcium efflux phenomenon in different tissues was examined. Calcium is known to play a major role in the regulation of secretion of neurotransmitters by nerve cells and of secretory proteins by endocrine to exocrine glandular cells (Schramm, 1967). To investigate whether increased calcium efflux from exposure to amplitude-modulated RFR is a common response of secretory cells, Albert et al. (1980) utilized rat pancreatic tissue slices in an experimental protocol essentially identical to that employed in the brain tissue studies of Blackman and similar to the protocols employed by Bawin and Adey, described above. Albert also measured changes in pulse-labeled secretory proteins. For the calcium-efflux study, data from 14 paired sets of pancreatic slices (irradiated or sham-irradiated slices paired with control slices) showed an 11% increase in calcium-ion efflux after 1-hour exposure, significant at the 3% level. Sham-irradiated tissue slices did not show statistically significant alteration in calcium efflux when compared with nonirradiated controls. Thus, 16-Hz

amplitude-modulated 147-MHz RFR at 2,000 microwatts/cm^2 increased calcium efflux from pancreatic tissue slices to approximately the same extent as that from neonate chick brain tissue incubated and exposed under similar conditions.

The pulse-labeled secretory protein study found that RFR had no effect on the release of these proteins from either normal or carbamylcholine-stimulated pancreatic tissue slices. However, because of physical constraints of the apparatus, the tissue slices could only be incubated in 1 ml of incubation medium, instead of the 15 ml used in other established in vitro secretion studies on pancreatic tissue slices. Therefore, the investigators believe that the conditions did not permit them to test most effectively for an RFR effect. When the incubation medium was increased to 15 ml, carbamylcholine resulted in the release of 30% of the pulse-labeled secretory protein. Albert et al. (1980) therefore suggested that the experimental conditions that promoted increased calcium efflux in their experiments, in the experiments of Blackman and colleagues, and in the experiments of Bawin and Adey and colleagues may not be optimal for testing the full potential effects of RFR. They also suggested that such RFR-induced calcium-ion efflux changes have little physiological significance.

In the studies already cited in this section, Bawin and Adey attributed the increase in release of calcium ion to an interaction between the amplitude-modulated RFR and specialized binding sites on the outside surface of plasma membranes of brain cells. Allis and Fromme (1979) pointed out that calcium is also transported across biological membranes by energy-dependent processes, and that sinusoidally modulated RFR may exert an effect on the function of membrane-bound enzyme systems mediating such transmembrane transportation. They therefore conducted experiments wherein specially prepared membrane-bound enzyme systems were irradiated with sinusoidally modulated RFR in a spectrophotometric apparatus in which enzyme activity was measured during irradiation. Cytochrome oxidase, a key enzyme that is located in the inner membrane of mitochondria, and adenosine triphosphatase from red-blood-cell membrane, which is involved in maintaining the sodium-potassium balance of the cell, were studied. The SAR was 26 W/kg for irradiation with 2,450-MHz RFR amplitude-modulated at 16, 30, 90, and 120 Hz. No statistically significant differences in enzyme activities were found between irradiated and control samples at any of the modulation frequencies for either enzyme system. However, as Allis and Fromme point out, these results are not definitive for several reasons: the membranes were tested for two functions under highly artifical conditions, e.g., the resting electrical potential across the membrane was not maintained in the in vitro preparation they employed; the exposure levels used were much higher than the "power window" demonstrated by the previously cited research above; and only the 16-Hz modulated frequency is within the "frequency window" claimed for nervous-tissue effects.

Although the calcium efflux phenomenon is of general interest because of the low average power densities involved, the phenomenon is not directly relevant to OTH-B, because the RFR therefrom is not amplitude modulated.

C.6.5.3 Blood-Brain Barrier Effects

The existence of a "blood-brain barrier" (BBB) in most regions of the brain has been established experimentally, although its specific morphology is still conjectural. This barrier normally provides high resistance to movements of large molecules (e.g., proteins or polypeptides) from the blood vessels into the surrounding brain tissue, presumably to protect the brain from invasion by various blood-borne pathogens and toxic substances. Several investigators have reported that low levels of RFR can increase the permeability of the BBB to certain substances of large molecular weight. However, others were unable to confirm such effects, and the subject remains controversial.

Rodzilsky and Olszewsky (1957) found that permeability changes in cerebral blood vessels could be induced by various non-RFR means, including those that produce heat necrosis. Sutton and co-workers (Sutton et al., 1973; Sutton and Carroll, 1979), who were interested in the use of RFR for selective hyperthermic treatment of brain tumors, determined the maximum temperatures and exposure durations that would not alter the integrity of the BBB in the rat. They used 2.45 GHz to induce hyperthermia, and horseradish peroxidase (HRP), a high-molecular-weight protein, as a tracer that is detectable both morphologically and quantitatively. Heads of rats were heated with RFR to a brain temperature of 40, 42, or 45 deg C. They found that BBB integrity was diminished, in orthonormic animals (37 deg C), by heating the brain to 45 deg C for 10 min, to 42 deg C for 15 min, and to 40 deg C for 60 min. The corresponding durations in rats precooled to 30 deg C were 15, 30, and 180 min, respectively.

Frey et al. (1975) exposed groups of anesthetized rats to pulsed or CW RFR at 1.2 GHz for 30 minutes. For the pulsed RFR, the pulse and average power densities were 2.1 and 0.2 mW/cm^2, respectively, and the power density of the CW RFR was 2.4 mW/cm^2. Sham-exposed rats were used as controls. After exposure or sham exposure, sodium fluorescein was injected into the femoral vein. Five minutes after injection, the blood of the rat was withdrawn, and the brain was removed, embedded in gelatin, refrigerated, and sectioned. The sections were viewed under ultraviolet light for fluorescence, the intensity of which was scored by the viewer. Greater fluorescence was reported for pulsed than for CW RFR, and some control specimens also exhibited slight fluorescence. The investigators regard these results as evidence that exposure to RFR alters the BBB.

Spackman et al. (1978) performed a similar investigation in mice, using fluorescein and several nonphysiological amino acids as test

substances. Groups of mice were exposed to sham, CW, or pulsed RFR at 918 MHz for 30 minutes. Average power densities of 2.5 and 33 mW/cm^2 were used in both the CW and pulsed modes. Also, some mice were exposed to CW RFR at 132 mW/cm^2. After exposure, the concentration of each test substance in the brain relative to the concentration of that substance in the blood plasma (the "specific concentration") was determined. A spectrofluorometer and an automatic amino acid analyzer were used to measure the concentrations of fluorescein and the test amino acids, respectively. The specific concentrations of all substances tested in the RFR-exposed animals were found to be in the same ranges as for the controls.

Subsequently, Spackman et al. (1979) used whole-body heating or intraperitoneal (i.p.) injection of glycerol, urea, metaraminol, or dimethyl sulfoxide (DMSO) as alternative agents to RFR exposure. They found that heating mice to 50 deg C in an incubator for 22 to 25 min caused no apparent increase in BBB permeability to the test substances. The same results were obtained for all of the injected agents except for DMSO, which produced a significant increase in BBB permeability relative to controls.

Albert et al. (1977) also used HRP as a tracer and reported regions of leakage in the microvasculature of the brains of Chinese hamsters exposed to 2.45-GHz CW RFR at 10 mW/cm^2 for 2 to 8 hours. In control animals, extravascular reaction product was found only in brain regions normally lacking a BBB.

In a later study, Albert (1979) exposed 52 animals (34 Chinese hamsters and 18 rats) to 2.8-GHz RFR for 2 hours at 10 mW/cm^2. Of these, 30 were euthanized immediately, 11 at 1 hour after exposure, and 11 at 2 hours after exposure. Twenty animals (12 hamsters and 8 rats) were sham-exposed. Leakage of HRP in some brain regions was reported for 17 of the 30 animals euthanized immediately after RFR exposure and for 4 of the 20 sham-exposed animals. Fewer areas of increased BBB permeability were evident for animals euthanized 1 hour after RFR exposure, and except for one rat, virtually no leakage of HRP was seen for the animals euthanized 2 hours after RFR exposure. These results indicate that decreased BBB permeability due to RFR exposure at levels that are insufficient to denature brain tissue is a reversible effect. (Albert suggests that such BBB changes may be clinically subacute and would probably cause no lasting ill effects.) However, the detection of increased BBB permeability in 4 of the 20 sham-exposed animals may be an indication that factors other than RFR in the experimental procedure could alter the BBB. One possible confounding point in the use of injected HRP as a tracer is the existence of endogenous peroxidase, the detection of which could yield false positive results.

Oscar and Hawkins (1977) reported changes in BBB permeability to D-mannitol due to exposure of rats to 1.3-GHz pulsed or CW RFR for 200 minutes at various average power densities. Permeability changes were measured by the Oldendorf (1970, 1971) technique; that is, 0.2 ml of a mixture of 14-C-labeled mannitol and tritiated water was injected

rapidly into each rat's carotid artery after exposure, the animal was euthanized 15 s later, and brain sections were dissected out and prepared for assays of radioactivity using a liquid scintillation counter. The ratio of counts of 14-C-labeled D-mannitol to counts of freely diffusible tritiated water in samples of brain tissue was normalized to a similar ratio for the injected solution. This normalized ratio, expressed as a percentage, is defined as the brain uptake index (BUI). Oscar and Hawkins found statistically significant changes in the BUI at average power densities less than 3 mW/cm^2. They also found that pulsed RFR could be either more or less effective in altering BBB permeability than CW RFR of the same average power density, depending on the specific pulse characteristics used. For pulses of long duration and high pulse power density but only a few pulses per second, mannitol permeation could be induced at average power densities as low as 30 microwatts/cm^2. Their results also indicated the possible existence of a power density "window;" i.e., permeability is not altered for power densities above or below the window.

Preston et al. (1978), using methods similar to those of Oscar and Hawkins, attempted to determine whether exposure to 2.45-GHz CW RFR increased BBB permeability to 14-C-labeled D-mannitol. They exposed rats to 0.1, 0.5, 1, or 10 mW/cm^2, with sham-exposed rats for controls, and found no evidence that RFR exposure increased the permeability of the BBB for mannitol.

Chang et al. (1978) used a technique involving 131-I-labeled albumin to investigate alterations of the BBB in dogs. The heads of dogs were exposed to various average power densities between 2 and 200 mW/cm^2. In general, no statistically significant differences were found between exposed and sham-exposed animals, but the number of animals used in this study was too small to ascribe a high level of statistical confidence.

Merritt et al. (1978) endeavored to replicate the studies of Frey et al. (1975) and Oscar and Hawkins (1977). They exposed rats for 30 min to 1.2-GHz pulsed RFR at peak power densities in the range from 2 to 75 mW/cm^2 and 0.5 duty cycle, corresponding to average power densities of 1 to 38 mW/cm^2, or for 35 min to 1.3-GHz pulsed or CW RFR at average power densities in the range from 0.1 to 20 mW/cm^2. In addition to examining brain slices under ultraviolet light for fluorescein transfer and under white light for Evans blue dye transfer across the BBB, they chemically analyzed various brain regions for fluorescein content. They also measured the brain uptake of 14-C-labeled D-mannitol and determined the BUI values. To validate these detection methods, they used hypertonic urea, known to alter the BBB, as an alternative agent to RFR. Last, sham-exposed rats were heated for 30 min in a 43 deg C oven to appproximate the hyperthermia obtained at 38 mW/cm^2. In their examination of brain slices, they found no evidence of fluorescein or Evans blue dye transfer across the BBB of RFR-exposed rats, whereas penetration of the BBB was apparent for

rats treated with urea instead of RFR. The analyses of fluorescein content corroborated these findings. However, fluorescein uptake was higher for the sham-exposed rats that were heated in the oven, an indication that hyperthermia of the brain is necessary to alter BBB permeability. In the 14-C-mannitol study of the various brain regions, there were no significant differences in BUI between RFR- and sham-exposed rats, whereas BUI changes were evident for rats treated with urea. Also, the results showed no evidence of the power density window reported by Oscar and Hawkins.

The use of urea as a positive control (alternative agent to RFR) by Merritt et al. adds considerable weight to their negative findings concerning RFR as a possible BBB-altering agent. However, a basic uncertainty in this and most earlier research on this topic is whether significant artifacts are introduced by the kinds of biological techniques used (e.g., the effects of anesthesia in many animals on the results obtained are difficult to ascertain). Several investigators have indicated that exposure to RFR may alter the size of vascular and extravascular volumes and cerebral blood flow rate, thereby yielding changes in the BUI that are not necessarily related to BBB permeability alterations. Blasberg (1979) reviewed many of the methods previously used for investigating BBB changes and the problems associated with these methods. Rapoport et al. (1979) have developed a method for measuring cerebrovascular permeability to 14-C-labeled sucrose that yields results that are independent of cerebral blood flow rate. Oscar et al. (1979) have confirmed experimentally that local cerebral blood flow is increased in the rat brain by exposure to pulsed RFR at either 1 or 15 mW/cm^2 average power density.

Recent findings such as those mentioned above indicate that little quantitative confidence can be placed in the results of early experiments on RFR-induced BBB alterations. Qualitatively, it is clear that hyperthermic levels of RFR can alter the permeability of the BBB. It is also possible that exposure at average power densities of the order of 10 mW/cm^2 may result in randomly distributed, clinically subacute, reversible alterations. However, additional research using current or improved methodology is necessary to ascertain whether chronic exposure to nonhyperthermic levels of RFR affects the BBB. Recent reviews of this topic were published by Justesen (1980) and Albert (1979).

At present, there is no evidence that alterations of the permeability of the BBB would result from exposure to the levels of RFR from OTH-B outside the exclusion fence.

C.6.5.4 Histopathology of the Central Nervous System (CNS)

Tolgskaya and Gordon (1973) reported a number of effects of RFR (frequencies 500 kHz to 100 GHz) on approximately 646 animals, predominantly rats. Pathological effects they reported for high-intensity (20 to 240 mW/cm^2) RFR in their so-called decimeter band (500 MHz to 1 GHz) included multiple perivascular hemorrhages in the brain and other organs, degeneration of apical dendrites in the cortex, cloudy swelling of cytoplasm, cytoplasmic shrinkage, formation of vacuoles, unevenness of staining, disappearance of cytoplasmic structures, fatty degeneration, decrease in ribonucleoprotein, and occasional karyocytolysis. The intensities of exposure were capable of causing death of the animals (clinical signs of hyperthermia, temperature increases up to 42 deg to 45 deg C) in several minutes to several hours. Photographs of the exposure arrangement show multiple animal exposures at the same time in a room appearing not to have RFR-absorbing material on the walls. It is likely that the specific absorption rates (SARs) for individual animals under these conditions varied widely, and that all effects were clearly thermal in nature.

Exposures referred to as "low-intensity" were also performed. The authors define threshold field intensities for nonthermal effects ("intensity not raising body temperature") for decimeter microwaves as 40 mW/cm^2 (Tolgskaya and Gordon, 1973, Table 3, p. 56). Exposures were generally at or slightly below 10 mW/cm^2 for 60 min daily for 10 months. Investigation of the animals by ordinary morphological methods revealed practically no vascular disorders in the nervous system. "Delicate elective neurohistological methods" (unspecified) showed disappearance of spines from cortical dendrites, the appearance of beading and irregular thickening of dendrites, swelling of cytoplasm of individual cells (with appearance of vacuoles) in the basal ganglia and hypothalamus, focal and diffuse proliferation of microglial cells, with microglial processes showing initial signs of degeneration.

Many of these "low-intensity" effects are similar to those described for the high-intensity exposures. In view of the exposure levels (approximately 10 mW/cm^2), the previously described exposure arrangement, and the knowledge of the possibility of localized regions of high SAR, it seems likely that the described effects (more subtle than those of frank hyperthermia) were also thermal in origin.

Albert and DeSantis (1975) reported changes in the hypothalamus and subthalamus of Chinese hamsters exposed to 2.45-GHz RFR at either 50 mW/cm^2 for durations from 30 min to 24 hr, or 25 mW/cm^2 for 14 hr/day for 22 days. Changes were not evident in the hippocampus, cerebellum, thalamus, or spinal cord ventral horn. In the discussion printed after the paper by Albert and DeSantis (1975), Guy pointed out that his laboratory had measured mean SARs as high as 4 W/kg per incident mW/cm^2 in animals of similar size. Peak SARs could have reached 40 to 200 W/kg in selected brain regions of the animals studied

by Albert and DeSantis; this range far exceeds that normally used for diathermy treatment in 20-min exposures of patients. Rectal temperature measurement would not necessarily reflect such high SARs in localized areas.

Albert and DeSantis (1976), also studied CNS histological effects in 60 Chinese hamsters exposed to 1.7-GHz RFR at power densities of 10 and 25 mW/cm^2. Cytopathology was observed after 30 to 120 min of exposure in hypothalamic and subthalamic areas, but not in other areas. These observed effects were also likely to have been thermal in origin, for the reasons previously mentioned.

Merritt and Frazer (1975) conducted a study to determine whether HF (19 MHz) RFR altered the whole brain level of certain neurotransmitters in mouse brain. The neurochemicals they studied were serotonin and its metabolite, 5-hydroxyindole acetic acid; dopamine and its metabolite, homovanillic acid; and norepinephrine. Male adult Swiss Webster mice were housed individually in plastic cages and exposed in groups of 5 to 14 to either predominantly E- or predominantly H-fields in a near-field synthesizer. "E-field" exposure was at 6 kV/m with an "impedance" of approximately 940 ohms. "H-field" exposure was at 41 A/m with an "impedance" of approximately 49 ohms. Exposures were for 10 min. These exposures did not produce measurable rises in rectal temperature. Two sets of control animals were sham-exposed in the near-field synthesizer. Fifteen minutes after completion of exposure (or sham exposure), one set of controls and all sets of exposed animals were euthanized very rapidly by high power-density microwave inactivation of brain enzymes in a specially modified microwave oven plus waveguide system. The second set of controls was euthanized 15 minutes after exposure by cervical dislocation, and their brains were rapidly dissected out and immediately frozen in liquid nitrogen for enzyme inactivation. The enzyme-inactivated brains were then assayed for the five neurochemicals mentioned above by a modification of a standard spectrofluorometric technique.

Results of the assay indicated that there were no statistically significant differences in whole-brain-averaged values between the microwave inactivation controls and either E-field-exposed or H-field-exposed animals for any of the neurotransmitters or their metabolites. The microwave-inactivated controls did show a statistically significant difference (microwave control values higher) for all neurochemicals except homovanillic acid when compared with the conventional controls. Because of the rapidity of the microwave enzyme inactivation technique, this probably indicates rapid turnover of the neurotransmitters under study. Frequency scaling comparisons between human and mouse at 19 MHz indicates that for mouse exposure, approximately 30-40 times more incident power density is required to give the same whole-body average SAR as in a human. Therefore, for humans, short-duration E-field exposure up to approximately 150 V/m, or H-field exposure up to approximately 1 A/m at 19 MHz may be expected to have no effects on serotonin, 5-hydroxyindole acetic acid, dopamine, homovanillic acid, or norepinephrine in the brain.

In summary, RFR can cause observable histopathological changes in the CNS of animals, but it appears that these changes are thermal in nature. There is no evidence that such changes would be caused by exposure to the RFR from OTH-B outside the exclusion fence.

C.6.5.5 EEG Studies

Many studies have been conducted on the effects on the electroencephalogram (EEG) and/or evoked responses (ERs) of animals exposed to RFR. Some of these have been performed with metal electrodes either implanted in the brain or attached to the scalp during exposure. Johnson and Guy (1972) pointed out that such metallic electrodes grossly perturb the fields and produce greatly enhanced absorption of energy (i.e., field enhancement) in the vicinity of the electrodes. Examples of the magnitude of this enhancement were provided in a National Academy of Sciences publication (NAS, 1979). For an implanted insulated wire, with the end of the conductor in direct contact with the tissue and a length-to-radius ratio of the wire of 100:1, the enhancement factor for SAR is of the order of 100,000. Such increases probably result in highly localized transient heating in the immediate vicinity of conductive implants in tissue exposed to microwave fields with time-averaged intensities greater than 1 microwatt/cm^2. The greatly enhanced fields themselves are also likely to cause artifacts in nervous system tissue function in the volume immediately around the electrode because of the sensitivity of such tissue to electrical stimulation. Such artifacts are not to be confused with the recording artifact that is produced by pickup of fields by the electrodes and leads during the recording of EEGs or ERs while the animal is being exposed. Recording artifact is usually removed by appropriate filtering. Field enhancement artifact is not altered by such filtering techniques. Note also that many EEG studies are performed on heavily sedated animals, with barbiturates as the usual drugs. Hence the responses reported do not necessarily reflect those that would be expected in normal alert animals.

Tyazhelov et al. (1977) discussed these problems and pointed out that even for the coaxial electrode developed by Frey et al. (1968), diffraction of electromagnetic waves is still a major source of error because of the electrode's metallic nature and large dimensions. Tyazhelov solved the problems by developing electrodes of high linear resistance (greater than 100 kilohms/m) and properly filtering the recorded signal. This report indicates an awareness in the USSR that questions may be raised about the validity of data and conclusions from many experiments involving animals with indwelling electrodes, both in the USSR and the United States.

Bruce-Wolfe and Justesen (1979) investigated the effects of RFR-induced hyperthermia on the visually evoked electrocortical response (VER) in five female guinea pigs. The VERs were recorded for animals after exposure to modulated 2.45-GHz RFR in a multimode, mode-stirred cavity for durations ranging from 4 to 15 min. Such exposures raised

rectal temperature to as high as 43 deg C and brain temperature to as high as 41 deg C. The mean latency from onset of photic stimulation to the N_1 peak diminished from 42.8 to 37.7 ms for cortical temperature increases of 37.0 to 40.5 deg C. For a cortical temperature above 41.5 deg C, the VER became highly variable, and above 43.0 deg C the animals died. The authors recognized the possibility of local brain damage resulting from the use of implanted metal electrodes and stainless-steel screws, and stated that this aspect was being explored in further studies in the rat and guinea pig.

Takashima et al. (1979) studied the effects of modulated RFR fields on the EEGs of rabbits. Male rabbits were exposed for 2 hours a day for 6 weeks to 1- to 10-MHz RFR modulated at 15 Hz. Although the authors claimed an enhancement of low-frequency components and an increase in high-frequency activity after 3 weeks, the data presented do not support this conclusion, and the authors themselves state that the results presented are still incomplete. They did note that for acute irradiation, "enhanced slow waves and unusually low high-frequency activities were due to the local field created by the presence of the (recording) metal electrodes in the cranial cavity."

Dumanskij and Shandala (1974) reported changes in the biocurrents in the brain cortex in rabbits after 60 days of exposure to RFR (50 MHz, 2.45 GHz, or 10 GHz). Changes (vaguely specified as "an increase in the rhythm of slow waves and a decrease in the rhythm of intermediate and fast waves") were described at 10 and 1.9 microwatts/cm^2, but not at 0.01 microwatts/cm^2. Although the rather sketchy nature of their description precludes any definitive evaluation of these results, it appears that the use of indwelling electrodes may have contributed artifacts, as described above.

In a more recent presentation, Shandala et al. (1979) reported on observations of rabbits with implanted EEG electrodes, and again claimed quite variable, but statistically significant, EEG changes at 10 microwatts/cm^2 exposures (2.375 GHz) for 7 hours/day for 1 month. The same questions about the possibility that implanted electrodes caused artifacts may be raised.

Goldstein and Cisko (1974) studied the EEGs of sedated rabbits to determine whether RFR exposure would evoke arousal. They used 9.3-GHz RFR at 0.7 to 2.8 mW/cm^2. The EEG of each rabbit was recorded for about 1 hr. After the first 10 min, the rabbit was sedated with sodium pentobarbital. Five minutes later, the rabbit was exposed or sham-exposed to the RFR for 5 min. The EEGs showed no arousal during RFR exposure but indicated alternations of arousal and sedation characteristics starting 3 to 12 min after exposure. However, control animals also exhibited alternations having shorter arousal durations, rendering interpretation of these results difficult. These investigators were aware of the potential problem of metals in the pathway of the RFR and claimed to have mitigated it by using thin

(0.01-inch), insulated, implanted stainless steel electrodes. It is unlikely that this reduced the artifacts significantly, if at all, because the thickness of the metal was still much greater than the metallic "skin depth," and also because using thinner electrodes actually increases the length-to-thickness ratio and increases the field enhancement (NAS, 1979). They also stated that "under everyday conditions, the EEG patterns of rabbits are quite variable. The animals oscillate between sedation and arousal unpredictably." This variability is another potential source of error in any experiments on the EEG of rabbits.

Chou et al. (1978) used implanted carbon electrodes to avoid the artifactual problems associated with metal ones. Two groups of rabbits (six animals/group, three males, three females) were exposed to 2.45-GHz, 1.5 mW/cm^2 RFR for 2 hr daily for 3 months. One group received CW, the other pulsed RFR (10 microseconds, 100 pps, 1.5 W/cm^2 pulse power density). A similar group of six animals was sham-exposed. No significant differences in EEG and evoked potentials were observed at the end of 3 months.

Kaplan et al (1980) reported that, from the beginning of the second trimester of pregnancy, 33 squirrel monkeys were exposed for 3 hr/day in special cavity/cage modules to 2.45-GHz pulsed RFR at whole-body mean SARs equivalent to those resulting from plane-wave exposure to 0.1, 1, and 10 mW/cm^2 and compared with a group of eight pregnant sham-exposed monkeys. Eighteen of the exposed mothers were exposed with their offspring for an additional 6 months after parturition, and then their offspring were exposed alone for another 6 months after weaning. No statistically significant differences were found between exposed and nonexposed adults nor between exposed and nonexposed offspring on resting EEG and photically driven EEG parameters. (No chronically attached or indwelling electrodes were used.)

Rosenstein (1976) exposed 1 group of 8 female rats to 10 mW/cm^2 at 425 MHz for 4 hr/day from the 12th day after breeding until parturition, and another group of 12 dams to 5 mW/cm^2 at 2.45-GHz for 4 hr/day from the 6th day after breeding until parturition. The offspring were then exposed for 92 days. Control groups with equal populations were used for each frequency. Evaluation of the EEGs and the visual ERs of the offspring at 140 days of age indicated no significant difference between the exposed and control groups. (Again, indwelling electrodes were not used.)

In summary, the use of indwelling metallic electrodes in studies on the effects of RFR on the EEG and/or evoked potentials may be questioned as a procedure likely to introduce artifactual effects in the preparation under study, as well as in the recordings themselves. These artifacts may be minimized by use of electrodes appropriately designed from high resistivity materials. Experiments in which such specially constructed electrodes were used, or in which electrodes were applied

after exposure, show no evidence of statistically significant differences in EEGs or evoked responses between control and RFR-exposed animals.

C.6.6 Effects on Behavior

The very large number and variety of behavioral studies in animals exposed to RFR make it impractical to present a detailed review of each one. The papers reviewed in this section were selected as representative of the types of behavioral studies that have been conducted, which have included studies of effects on reflex activity, RFR-perception studies, evaluations of effects of RFR on learning and on performance of trained tasks, studies of interactive effects of RFR and drugs on behavior, and investigations of behavioral thermoregulation.

Summaries of recent studies on behavioral effects being conducted under an ongoing cooperative program between the USSR and the United States on the biological effects of RFR are contained in two progress reports (Shandala et al., 1979; McRee et al., 1979). The Soviet studies (Shandala et al., 1979) evaluated the effects on rats of exposures at 10, 50, and 500 microwatts/cm^2 exposures for 1 to 3 months by measuring parameters giving "characteristics of the inborn forms of behavior (investigative behavior, feeding behavior, and aggressiveness), conditioned reflex activity (the rate of development of conditioned reflexes), effector and receptor behavioral reactions (locomotor activity, working capacity, and skin sensitivity to irritation by electricity)." For all of these parameters, the results presented showed differences between exposed and control animals after approximately 10 days of exposure. The Soviet investigators concluded that "even exposure of animals to 10 microwatts/cm^2 results in disturbance of various forms of ... behavior ... (which) makes it possible to assume the presence under these conditions of a general suppression effect of the radiation on the function of the central nervous system." Unfortunately, insufficient detail is provided in the report to assess the validity of these claims for effects at such low levels.

The U.S. studies (McRee et al., 1979) reported on related parameters under higher exposure power densities (0.5 to 30 mW/cm^2) for exposure durations that varied from acute (55 min) to chronic (92 days). Measurements of reflex development (startle response, righting reflex) and age of eye opening showed no differences between exposed and control animals for power densities up to 10 mW/cm^2 for the continuing exposure conditions. For the same subjects, no consistent effects on locomotor activity were found at 120 and 240 days of age.

In another study, effects of a single exposure (55 min or 15 hr) on performance of a fixed ratio schedule of reinforcement were studied. The results indicated that performance decreased at power densities from 5 mW/cm^2 and up, but not at 0.5 or 1 mW/cm^2. Conflicts between

Soviet claims of effects at low (0.5 mW/cm^2 and lower) power densities under long-term exposure conditions and the absence of similar effects in the same power density range in the studies of United States researchers recur frequently in the RFR bioeffects literature.

The RFR hearing effect discussed in Section C.6.5.1 is, by definition, perception of RFR. Other studies of modulated RFR have been conducted to determine whether perception can serve as a behavioral cue.

King et al. (1971) showed that 2.45-GHz RFR, modulated at 60 and 12 Hz, could serve as a cue to warn rats of impending electrical shock. The effect had a threshold of 1.2 to 2.4 mW/g. Because the study was conducted in a cavity system, plane-wave power densities were not available; however, a reasonable estimate is that the power-density threshold would be between 2.5 and 6 mW/cm^2.

Frey and Feld (1975) showed that rats exposed to 1.2 GHz pulsed at 100 to 1,000 pps would tend to avoid the radiation by moving into an RF-shielded area. The authors interpreted this behavior as indicating that the RFR produced a noxious stimulus, but the noxious stimulus was probably the RFR hearing effect. This interpretation is substantiated by the observation of avoidance behavior at 0.6 and 0.2 mW/cm^2 for the 100 and 1000 pps exposures, respectively, although exposure to 2.4 mW/cm^2 CW RFR did not produce any avoidance behavior.

Several other RFR-perception studies were designed to determine whether animals would avoid CW RFR as a noxious stimulus. Monahan and Ho (1977) observed that mice exposed for various periods to 2.45-GHz RFR at various power densities and ambient temperatures were able to orient themselves to reduce the percentage of microwave energy absorbed. A threshold power level was apparent; the effect would occur at ambient temperatures of 20 and 24 deg C, but not at 30 and 35 deg C.

In a subsequent study, Gage et al. (1979a) observed rats and mice by closed-circuit TV during exposure to 2.45-GHz RFR and failed to observe orientation effects. However, comparison of power levels and ambient temperatures in the two experiments suggests that the power density and ambient temperatures employed by Gage et al. may have been too low to produce the orientation behavior.

Studies specifically designed to examine thermoregulatory behavior in the presence of 2.45-GHz RFR were performed with rats by Stern et al. (1979), and with squirrel monkeys by Adair and Adams (1980a). For the rats, behaviorally significant levels of heating (as determined from alterations in the rate at which the animals turned on an infrared heating lamp while they were in a cold environment) occurred at power densities ranging from 5 to 20 mW/cm^2. For the squirrel monkeys, alterations in thermoregulatory behavior (controlling the environmental temperature by adjusting the rate of warm air flowing into a cold chamber) occurred at a threshold of approximately 6 to 8 mW/cm^2. Both

of these results were in highly trained animals and occurred in the absence of measurable changes in other parameters such as colonic, rectal, or skin temperatures.

A second approach to the question of RFR avoidance (Monahan and Henton, 1979) involved exposing mice to 2.45-GHz RFR at an average SAR of 45 mW/g (90 to 100 mW/cm^2), coupled with a sonic cue. The mice learned to turn off the RFR by interrupting a beam of light; this was interpreted as escape behavior.

A third approach involved pairing exposure to RFR with consumption of sucrose solution and subsequently testing for avoidance of the sucrose. An earlier test (Monahan and Henton, 1977) produced no evidence that the sucrose was associated with a noxious experience. In a subsequent test (Sessions, 1979), saccharin (instead of sucrose) was paired with RFR exposure of rats. The animals developed aversion to the saccharin (indicating association with a noxious experience) at a power density of 41 mW/cm^2 or greater, but not at lower levels.

The results cited above are interpreted by the authors as indicating that RFR is a noxious or unpleasant stimulus to the animal. However, the orientation behavior observed by Monahan and Ho (1977) appears to be related to thermoregulatory behavior of the animals, and the saccharin aversion appears to require relatively high power densities of the RFR. The escape by interrupting the beam of light (Monahan and Henton, 1979) also involved relatively high power densities, and in addition, appeared to require the coupled sonic cue. Grove et al. (1979) observed that rats exposed to nearly lethal levels of RFR in the absence of other cues (e.g., sonic cues) made no attempt to escape, even though the means of escape were readily available. Overall, it can be concluded that pulsed or otherwise modulated RFR can be perceived readily by animals at moderate-to-low power densities, but that CW RFR is, at best, an extremely feeble perceptual cue.

Many studies have been conducted on the effects of RFR on the performance of trained tasks. Animals studied have been rats, rhesus monkeys, and squirrel monkeys. Acute exposures at power-density levels ranging from 10 to greater than 100 mW/cm^2 (Sanza and de Lorge, 1977; de Lorge, 1979; D'Andrea et al., 1977; Lin et al., 1977; Scholl and Allen, 1979; de Lorge and Ezell, 1979; McAfee et al., 1979) resulted in somewhat inconsistent results. The overall conclusion is that RFR will suppress performance of learned tasks, but that the effect depends on power density, duration of exposure, animal species, and the demand characteristics of the behavior. The studies of de Lorge and Ezell (1979) indicate that suppression of learned behavior tasks by exposure to high levels of RFR depends on the amount and distribution of energy absorbed by the animal.

Chronic exposure to RFR has also been reported to disrupt learned behavior in animals. Lobanova (1974) reported a weakening of

conditioned reflexes in rabbits and rats, as shown by increased latency or absence of response and failure to recognize the conditioned stimulus. Power-density levels in her studies were 1 to 10 mW/cm^2.

Mitchell et al. (1977) reported that rats showed an increase in locomotor activity and a disturbance of differential responding to operant behavior over a 22-week exposure at 2.3 mW/g (5 to 6 mW/cm^2). Lebovitz and Seaman (1979), however, found no disturbance in lever-pressing performance in rats chronically exposed at up to 2.6 mW/g.

Studies of the effect of RFR on learning are more recent and fewer. Schrot et al. (1979) investigated the effects of RFR on the ability of rats to learn a novel sequence of responses to obtain food reinforcement. Decrements of learning occurred at power densities of 5 and 10 mW/cm^2, but not at 1 mW/cm^2 or less. Gage et al. (1979b) exposed rats daily for 4 hr to 50 mW/cm^2 from day 6 of gestation until the age of 126 days, and found no effect of the RFR on the learning of two tasks during the last 2 weeks of exposure.

Several behavioral studies of interaction of RFR and drugs that affect the CNS have been conducted (Monahan and Henton, 1979; Thomas and Maitland, 1979; Thomas et al., 1979; Maitland, 1979). In the first study, chlordiazepoxide was found to interfere with avoidance responses to 2.45-GHz CW RFR at a mean SAR of 45 W/kg, but chlorpromazine and d-amphetamine gave variable results. In the other studies, the effect of a drug on animal behavior was tested, and then the effect of the drug and RFR together was tested. Pulsed 2.45-GHz RFR at an average power density of 1 mW/cm^2 (SAR of 0.2 W/kg) was found to enhance the effects of dextroamphetamine, chlordiazepoxide, and pentobarbital. An interesting aspect of these studies, taken together, is that the drugs have pharmacologically different and opposite properties. Dextroamphetamine is a CNS stimulant, whereas chlordiazepoxide and pentobarbital are CNS depressants, but the RFR enhances the effect in either case.

Thomas et al. (1980) extended their work on chlordiazepoxide to include the interactive effects between RFR (pulsed 2.8 GHz) and chlorpromazine, and between RFR and diazepam. In contrast to the findings with chlordiazepoxide, exposure to 1 mW/cm^2 of RFR in conjunction with either chlorpromazine or diazepam did not produce any alterations in the behavioral dose-effect functions. Diazepam and chlordiazepoxide are in the same class of drugs, whereas chlorpromazine is in a different class. Therefore, pharmacological activity or classification is not sufficient to predict synergistic or antagonistic effects with low-level RFR exposure.

In summary, some of the behavioral studies seem to have originated from studies in the USSR claiming that RFR had direct effects on the CNS at low power densities. The association is discussed by King et al. (1971). Evidence to support this claim from neurophysiological studies

in the United States is meager, and the behavioral evidence also does not generally support the claim. The studies on RFR as a noxious stimulus do not show that the animals can perceive RFR as such. The radiation avoidance observed appears to be part of the thermoregulatory behavior of animals; when the environment is cold, animals will use RFR as a source of warmth (Stern et al., 1979). In addition, Adair and Adams (1980b) showed that RFR enhanced dermal vasodilation in the squirrel monkey (a thermoregulatory response). The effect appeared to be mediated by the CNS, but a minimum of 8 mW/cm^2 was required to elicit the response. Disruption of performance or learning appears to have rather high power-density thresholds. Interaction of RFR with drugs affecting the CNS appears to be the most sensitive behavioral response to RFR, but even these studies do not prove a direct effect of RFR on the CNS. Overall, the behavioral studies do not indicate a special effect of RFR on the nervous system, and the mechanism of most of the results remains conjectural.

In conclusion, there is no evidence that exposure of humans solely to the RFR from OTH-B outside the exclusion fence will alter their behavior. There is some evidence for synergistic effects on rats of certain drugs and RFR at average power densities (for 2.45 and 2.8 GHz) of the order of 1 mW/cm^2, the approximate level at the exclusion fence. However, there is no evidence that such synergistic effects would occur in humans in the nearby communities, where the average power densities are much lower.

C.6.7 Endocrinological Effects

Exposure of animals to RFR has produced somewhat inconsistent effects on the endocrine system of mammals. In general, the effects produced appear to be related to either the heat load associated with the RFR or the stress induced in the animals by the RFR and, possibly, other experimental circumstances. Some effects also appear to be related to alteration of the circadian rhythm by RFR. There do not appear to be any effects clearly demonstrated to be associated with nonthermogenic stimulation of the endocrine system or the associated parts of the CNS.

Magin et al. (1977a, 1977b) surgically exposed the two thyroid glands of dogs (under anesthesia) and used a diathermy unit and special applicator to irradiate one gland *in vivo* with 2.45-GHz RFR for 2 hr at 72, 162, or 236 mW/cm^2. The corresponding SARs in the gland were 58, 131, and 190 W/kg, and the resulting temperatures therein were about 39, 41, and 45 deg C. The other thyroid gland served as control. The release rate of the hormone thyroxine (T-4) into the blood was measured for both glands and was found to be higher by factors of 150, 350, and 1000%, respectively, for the gland exposed to the RFR. In addition, the blood flow rate in that gland was higher by 140 and 170% for temperatures of 41 and 45 deg C, respectively.

Lu et al. (1977) subjected rats first to a 2-week "gentling" period, and then sham-exposed or exposed them to 2.45-GHz RFR at 1, 5, 10, or 20 mW/cm^2 for 1, 2, 4, or 8 hr starting at the same hour on the same day of the week. After treatment, each rat was decapitated, blood was collected, and body mass and rectal temperature were measured. In addition, the pituitary, adrenal, and thyroid glands were weighed. The levels in blood serum of corticosteroid (CS), thyroxine (T-4), and growth hormone (GH) were assayed.

For the sham-exposed rats, mean rectal temperatures increased with exposure duration, an effect ascribed to circadian rhythmicity. The rectal temperatures of the RFR-exposed rats varied in an inconsistent manner. For example, for the 1-hr exposures, increases in mean temperature were noted for the groups exposed at 5 and 20 mW/cm^2, but not for those exposed at 1 and 10 mW/cm^2. The CS level increased with exposure duration for the sham-exposed rats and was correlated with the rectal temperature increase. Increases of CS level occurred in the RFR-exposed rats but were not significantly correlated with rectal temperature. The only significant changes in T-4 level were an increase for 4-hr exposure at 1 mW/cm^2 and decreases for 4-hr and 8-hr exposures at 20 mW/cm^2. No significant changes in GH level or in body mass or pituitary mass due to RFR exposure were noted. Several statistically significant alterations of thyroid and adrenal masses were observed, but no obvious pattern related to power density, exposure duration, or circadian rhythmicity was apparent. In view of the large variations in values for each endpoint in the rats sham-exposed for various durations (which presumably resulted from unknown differences in residual stress reactions after gentling, as well as circadian variations), it is difficult to discern any clear-cut effects ascribable to RFR exposure per se in these studies.

In a later study reported in an abstract (Lu et al., 1979), acclimated rats were sham-exposed or exposed to the same frequency for 4 hr at 0.1, 1, 10, 25, or 40 mW/cm^2, and the CS levels were assayed. The results showed decreases for 0.1 and 1 mW/cm^2, no significant changes for 10 and 25 mW/cm^2, and increases at 40 mW/cm^2. The authors ascribed the decreases at the lower power densities to RFR-induced circadian rhythm changes and the increases at the higher power density to RFR-induced stress in the animals.

In an abstract, Travers and Vetter (1978) reported exposing rats to 2.45-GHz RFR at 0, 4, or 8 mW/cm^2, 8 hr/day for 0, 7, 14, or 21 days. At 8 mW/cm^2, significant, highly correlated decreases in thyroxine and thyroid-stimulating hormone (TSH), as well as changes in several serum-protein levels, were observed; these results suggest that the depressed thyroid activity results from decreased TSH secretion by the hypothalamus in response to RFR exposure.

Czerski et al. (1974) presented evidence that exposure to RFR could alter circadian rhythms. They exposed mice to 2.95-GHz RFR at

0.5 mW/cm^2 for 4 hr in either the morning or the evening, and detected shifts in amplitude and phase of the circadian rhythm of mitosis (cell division) of precursor or "stem" cells in the bone marrow that differentiate and mature into various types of cells involved in immunologic functions.

Lotz and Michaelson (1978) "gentled" rats for 2 weeks and then exposed groups of 4 rats each to 2.45-GHz RFR for 30 or 60 min at power densities in the range from 0 (sham) to 60 mW/cm^2 or for 120 min at 0 to 40 mW/cm^2. (The mean SARs were 0.16 W/kg per mW/cm^2.) Plots of colonic temperature versus exposure duration at the various power densities showed a small but statistically significant temperature increase after 30-min exposure at 13 mW/cm^2; exposures for the same duration to higher power densities produced temperature increases approximately proportional to the power density. Plasma corticosterone levels were rather variable. At each power density, increases in mean level with exposure duration were discernible, but the results were not significantly different from baseline values for durations of up to 120 min at 13 mW/cm^2, up to 60 min at 20 mW/cm^2, and for 30 min at 30 mW/cm^2; these results are indicative of a threshold pattern of response. All other increases were significant. The increases in corticosterone level were highly correlated with the increases in colonic temperature. Estimates of the threshold SARs were 4.8 to 8.0 W/kg for 60-min exposure and 2.4 to 3.2 W/kg for 120-min exposure, with the latter range being somewhat less than half the resting metabolic rate for the rat. A major point demonstrated in this investigation is the necessity for gentling the rats and also equilibrating them for at least 3 hr prior to RFR- or sham-exposure, to minimize the non-RFR stresses imposed by the experimental situation.

Growth-hormone (GH) levels for similarly treated rats were measured by Lotz et al. (1977). For 30- and 60-min exposures, GH levels were lower than control values only at 50 and 60 mW/cm^2. For 120-min exposures, the GH levels were lower than those of controls at 13 mW/cm^2, and [illegible] reased progressively with increasing power density.

Lotz and Michaelson (1977) surgically removed the hypophysis (pituitary) of rats and exposed the rats to 2.45-GHz RFR for 60 min at [illegible]50, or 70 mW/cm^2 (mean SARs of 8.0, 9.6, or 11.2 W/kg). Assays of [illegible]ma samples for corticosterone indicated the absence of the increases [illegible]nd for intact and sham-hypophysectomized rats that were [illegible]60 min at 60 mW/cm^2. In addition, intact rats were [illegible]dexamethasone before exposure to RFR. For exposures at [illegible] min, the increases in corticosterone level found in [illegible] rats exposed to the RFR were absent. These results [illegible] adrenal gland is not primarily stimulated by exposure [illegible] mediated secondarily by ACTH secreted by the pituitary

Mikolajczak (1976, 1974) exposed male rats to 2.9-GHz RFR at 10 mW/cm^2 for 6 hr/day, 6 days/week over 6 weeks. Control rats were sham-exposed. Extracts of the pituitary glands of these rats were then assayed for follicle stimulating hormone (FSH) and growth hormone (GH) in female rats that had been hypophysectomized, and for luteinizing hormone (LH) in hypophysectomized male rats. No statistically significant differences in levels of FSH or GH between RFR- and sham-exposed animals were found. However, significantly higher levels of LH were reported for the RFR-exposed rats.

Because of the known sensitivity of the testes to heat, several investigations of the effects of RFR on gonadal function have been conducted. Prausnitz and Susskind (1962) exposed mice to 9.27-GHz RFR at 100 mW/cm^2 for 4.5 min/day (which increased mean body temperatures by 3.3 deg C) for 5 days/week over 59 weeks. Testicular degeneration was found in 40% of the RFR-exposed and in 8% of the control mice that had died during the course of the experiment. More recently, Cairnie and Harding (1979) reported that exposure of mice to 2.45-GHz RFR at 20 to 32 mW/cm^2 for 16 hr/day for 4 days had no effect on sperm count or percentages of abnormal sperm. No endocrinological measurements were reported for either study. Last, Lancranjan et al. (1975) reported that men occupationally exposed to RFR in the 3.6- to 10-GHz range at power densities of tens to hundreds of microwatts/cm^2 for 1 to 17 years (a mean of 8 years) showed slightly reduced sperm counts, but normal plasma levels of 17-ketosteroid and gonadotropic hormones.

In summary, although some of the effects of RFR exposure on the endocrine system appear to be relatively straightforward and predictable from physiological considerations, other, more subtle effects require further study, notably those related to the interactions among the pituitary, adrenal, thyroid, and hypothalamus glands and/or their secretions. Part of the problem in interpreting results appears to arise from uncertainties regarding stress mechanisms and accommodations thereto. Animals that are placed in novel situations are much more prone to exhibit stress responses than animals that have been adapted to the situation. However, there may be large variations in adaptation among animals in a given situation or among experimental situations in different laboratories. Moreover, the use of sham-treated controls may not always reduce the problem. A recent review of the effects of RFR on the endocrine system was published by Lu et al. (1980).

In conclusion, because the reported effects of RFR on the endocrine systems of animals are largely ascribable to increased thermal burdens, stresses engendered by the experimental situation, or both, there is no evidence that such effects would occur in humans exposed to the RFR from OTH-B at the power densities outside the exclusion fence.

C.6.8 Immunological Effects

The accumulation of reports to date indicates that RFR has quite definite effects on the immune system of mammals. Most of the reported effects were detected after exposure at power-density levels of about 10 mW/cm^2 and higher; a few have been detected following exposure to power densities as low as about 0.5 mW/cm^2; and in some cases, effects obtainable with the higher power-density range were not found at lower power densities. In most studies, the mechanisms for the effects seen were not investigated, and the various reports are somewhat inconsistent. Because of the complexity of the immune system and the variety of test procedures used, the representative studies discussed in this subsection are grouped into appropriate categories.

C.6.8.1 In Vitro Studies

In vitro studies are all related to the question of whether RFR can stimulate human or animal lymphocytes (a type of white blood cell of key importance in the immune system) to transform into lymphoblasts (mitotically active forms of lymphocytes) and undergo cell division (mitosis) when they are cultured outside of the body. Usually such cells are cultured in the presence of a mitogen (an agent, usually chemical) that stimulates blast transformation (i.e., lymphocyte to lymphoblast) and cell division.

One of the early studies was by Stodolnik-Baranska (1967, 1974), who cultured specimens of human lymphocytes, added the mitogen phytohemagglutinin (PHA) to one set of specimens, and exposed groups from both sets to 2.95-GHz pulsed RFR at an average power density of either 7 or 20 mW/cm^2 for various durations. The results for the PHA-stimulated cultures exposed a the higher power density showed no significant changes in percentages of blastoid forms, but there were significant decreases in percentages of lymphocytes and increases in mitotic index correlated with exposure duration (up to 40 min). The investigator indicated that similar results were obtained for exposures at the lower power density (for durations up to 4 hr), but presented no data. Regarding the cultures without PHA, she stated that RFR exposure induces the appearance of blastoid forms and microphage-like (scavenger) cells; she illustrated the point with one micrograph but gave no data.

As part of a larger study primarily involving exposure of animals, Czerski (1975) endeavored to repeat the experiments of Stodolnik-Baranska with human lymphocytes; he encountered difficulties in obtaining reproducible results. Czerski's 1975 work is discussed on p. 133 of Baranski and Czerski (1976), who implicated uncontrolled temperature increases in the specimens (which were not cooled during exposure). They stated that "exposures at power densities between 5 and 15 mW/cm^2, if continued till the moment when the temperature of the medium attained 38 deg C, could induce lymphoblastoid transformation; no such phenomenon could be obtained by exposure below 5 mW/cm^2."

Smialowicz (1976) prepared suspensions of mouse-spleen cells and exposed them to 2.45-GHz CW RFR at 10 mW/cm^2 (SAR of about 19 W/kg) for 1, 2, or 4 hr. Similar cell suspensions held at 37 deg C (without RFR exposure) for the same periods served as controls. After either treatment, specimens were cultured with or without one of four different mitogens (including PHA). Although changes in the extent of blast transformation with exposure duration were seen, there were no statistically significant differences between corresponding values for each duration from RFR-exposed and control specimens. This was true for the specimens not stimulated with mitogen and for those stimulated with any of the four mitogens. This investigator also measured the temperature and percentage viability of the specimens immediately after each treatment and found no significant differences in results between RFR-exposed and control specimens for each treatment period.

C.6.8.2 In Vivo Studies: Acute Exposures

In most in vivo investigations involving acute (i.e., short-duration) exposures, live animals were exposed one time for a period typically ranging from a few minutes to an hour at power densities high enough to produce substantial temperature increases in various tissues or organs or of the body as a whole. In general, the effects of such acute RFR exposure on the immune system appear to be stimulatory. The number of circulating lymphocytes in the blood increases, as does the ability of the immune system to manufacture antibodies to foreign substances. The number of cells involved in production of immune complement (a complicated series of interacting chemicals in the blood) also increases. The mechanisms of those effects are not completely understood, but in some cases they may be a secondary result of the stress induced in the animals by the RFR-produced heat or by other stresses, such as from handling.

Rotkovska and Vacek (1975) exposed mice to 2.45-GHz CW RFR at 10 mW/cm^2 for 5 min. Other mice were heated for 5 min in a ventilated chamber held at 43 deg C. Mean rectal temperatures increased by 2.3 and 2.5 deg C, respectively for the two groups. The leukocyte (white blood-cell) counts for the circulating blood of both groups increased, reaching maxima at 4 and 7 days after treatment for those exposed to RFR and at 4 days for those heated in the chamber. This effect was accompanied by increases in the numbers of nucleated cells in the spleen and the bone marrow of the femur over the same time period after treatment.

Krupp (1977a) exposed mice to 2.6-GHz RFR at 10, 15, or 20 mW/cm^2 for various durations. The mice were sensitized by inoculating them with sheep red blood cells (SRBC) at various times before and after exposure to the RFR, and the numbers of SRBC-antibody producing cells in the spleen were determined. The greatest increases in such cells (as compared with the values for sham-exposed, sensitized mice) were obtained when the mice were sensitized 4 hours after exposure to the

RFR. The effect was obtained when the exposure conditions produced a 3 deg C increase in rectal temperature. In addition, the effect could be elicited by the administration of cortisone, instead of RFR exposure, implying that the increases were adrenal-mediated responses to thermal stress.

Liburdy (1979) exposed mice to 26 MHz RFR at 80 mW/cm^2 (SAR of 5.6 W/kg) for 15 min. These exposures produced core (rectal) temperature increases of 2 to 3 deg C. For comparison, he heated mice in a dry-air oven at 63 deg C for the same period to obtain approximately the same increase in core temperature. (He also immersed mice in water at 41 deg C, but the rate of core temperature increase did not follow that for RFR exposure as well as the rate for the warm-air treatment.) Lymphopenia (diminution of the numbers of lymphocytes) and neutrophilia (increase in the proportion of neutrophils, which are white blood cells that perform different functions than lymphocytes) were evident in the RFR-exposed mice, effects that persisted for about 12 hr after exposure. The effects could be sustained and the recovery period prolonged by additional RFR exposures at 3-hr intervals. The effects were only slight for the mice heated in the oven. In addition, the effects were absent for mice exposed to 26-MHz RFR at 50 mW/cm^2 or to 5-MHz RFR at 800 mW/cm^2, both corresponding to 0.36 W/kg, or about one-sixteenth of the SAR used previously. Because heating may constitute a significant stress, this investigator determined the plasma corticoid levels, as a measure of such stress, after acute and chronic (longer-term, lower-level) RFR exposure, and found about a threefold increase in corticoid level relative to controls, whereas acute and chronic warm-air heating produced only modest, statistically insignificant increases.

There are two major classes of lymphocytes, which perform distinct immunologic functions both separately and in concert. Both classes originate from common immature lymphoid cells called stem cells. Stem cells that have infiltrated, differentiated, and matured in or under the influence of the thymus are called T lymphocytes or T cells. This type of cell interacts with a foreign body or antigen by direct cellular contact, to produce what is called the cellular immune response. Stem cells that are similarly derived from bone marrow are called B cells. Such cells interact with antigens by secreting specific proteins called immunoglobulins or antibodies and produce the humoral immune response.

Wiktor-Jedrzejczak et al. (1977) exposed mice to 2.45-GHz RFR in a waveguide at a mean SAR of 14 W/kg for either a single 30-min session or 3 such sessions, 1/day, 3 days apart. Control mice were sham-exposed. After exposure, the spleens were removed and tested for various effects of the RFR. First, the relative numbers of T and B lymphocytes in the spleens of the RFR-exposed mice were compared with those in the control mice. The results indicated that the total numbers of T cells were unaffected by either the single-session or triple-session exposures. However, the single exposures produced statistically significant

increases in the population of one subclass of B cells (complement-receptor-positive, or CR+) but not in another type of B cells (immunoglobulin-positive, or Ig+), whereas the triple exposures yielded increases in both types of B cells. Next, splenic cells from RFR- and sham-exposed mice were cultured after the addition of various T-cell-specific or B-cell-specific mitogens, and the numbers of cells undergoing blastic transformation were determined. Both the single and triple exposures yielded significant increases in blastic transformation of B cells but had insignificant corresponding effects on T cells. Last, groups of mice were inoculated with the antigen SBRC, which induces the production of antibodies by B cells provided that T cells are also present, or with another antigen (DNP-lys-Ficoll) that does not require the presence of T cells for antibody production by B cells. The mice were then given triple-session RFR exposures or sham exposures, after which their spleens were removed and assayed for antibody production. RFR-induced decreases in response to both antigens were observed, but only the difference for SRBC was statistically significant.

Taken together, the results of Wiktor-Jedrzejczak et al. (1977) indicate that acute exposures to the thermogenic levels of RFR used can have weak stimulatory effects on splenic B cells but none on T cells.

Regarding possible mechanisms, the observed increases in the numbers of CR+ B cells and of B cells undergoing blastic transformation could be manifestations of either RFR-induced proliferation of the B cell populations, which would be consonant with the findings of Stodolnik-Baranska (1967, 1974) and Czerski (1975), or RFR stimulation of immature B cells already present. In a subsequent paper, Wiktor-Jedrzejczak et al. (1980) presented data that support the latter hypothesis. In an abstract by Schlagel et al. (1979), coauthored by several of these investigators, results were presented suggesting that genetic factors may also play a role in lymphocyte response: mouse strains having the histocompatibility $H\text{-}2^k$ haplotype showed marked increases in CR+ cells due to RFR exposure, whereas those bearing the $H\text{-}2^b$ and $H\text{-}2^d$ haplotypes did not.

Huang and Mold (1980) exposed mice to 2.45-GHz RFR for 30 min/day at 5 to 15 mW/cm^2 (SARs of 3.7 to 11 W/kg) for 1 to 17 days, after which the spleens were removed and cells therefrom were cultured for 72 hours with or without the T-cell mitogens PHA or Concanavalin A (Con A) or the B-cell mitogen lipopolysaccharide (LPS). Tritiated thymidine, a radioactively labeled substance whose uptake is an indication of the DNA synthesis involved in cell proliferation, was added 4 hours before the end of the culturing period. The cells were then harvested and assayed for thymidine uptake. Plots of uptake versus exposure duration (made from the data in the authors' Table 1) showed biphasic or cyclical responses for cells from both mitogen-stimulated and nonstimulated cultures from the RFR-exposed mice. The investigators suggested that such cyclical fluctuations could account for the differences in results from various laboratories. However, similar plots for the sham-exposed

mice also show cyclical fluctuations, evidently resulting from factors other than RFR, such as circadian rhythms and estrus cycle changes in female mice; it is therefore impossible to ascertain the proliferative effects of RFR per se. In another part of the study, RFR exposure at 15 mW/cm^2 for 5 days (30 min/day) did not diminish the cytotoxic activity of lymphocytes on leukemic cells injected after, or concurrently with, the last exposure.

Huang et al. (1977) also exposed Chinese hamsters to RFR of the same frequency for 15 min/day on 5 consecutive days at power densities ranging from 0 to 45 mW/cm^2 (SARs from 0 to 20.7 W/kg). One hour after RFR (or sham) exposure, blood was drawn and cultured for 1 day if not mitogen-stimulated or for 3 days if stimulated with PHA. Cultures not stimulated with PHA exhibited a variation of the Transformation Index (percentage of transformed cells relative to the total number) with power density. The curve is in the shape of an inverted U; values peak at 30 mW/cm^2, and then gradually return to control values. Cell counts done when the blood was collected showed no net gain of lymphocytes from other sources, such as the lymph nodes or the spleen, and no significant changes in leukocyte differential counts; these counts support the contention that RFR does not cause lymphocytosis. For cultures stimulated with PHA, the mean value of Mitotic Index (percentage of cells in mitosis relative to the total number of lymphocytes) diminished from 3% for controls to about 0.04% and 0.05% for the 30 and 45 mW/cm^2 exposure groups, respectively.

C.6.8.3 In Vivo Studies: Chronic Exposures

Czerski (1975) exposed 100 mice to pulsed 2.95-GHz RFR at an average power density of 0.5 mW/cm^2 for 2 hr/day, 6 days/week over 6 weeks, and another 100 mice over 12 weeks. After exposure, these mice were immunized with SRBC. For controls, 100 unexposed mice were immunized and 2 other groups of 100 each were exposed for 6 and 12 weeks each but not immunized. On days 4, 6, 8, 12, and 20 after such treatments, the percentages of lymphoblasts and plasmocytes in suspensions of lymph-node cells and the numbers of antibody-forming cells were determined for subgroups of 5 mice each. For all 3 immunized groups (2 RFR-exposed and 1 unexposed), the percentage of blast cells peaked on day 6 and diminished to baseline values by day 20. The smallest maximum was for the unexposed group, the next larger maximum was for the group exposed for 12 weeks, and the highest maximum was for the 6-week-exposure group. Smaller peaks on day 6 were seen for the 2 nonimmunized RFR-exposed groups; again the higher value was for the group exposed for 6 weeks. Qualitatively similar results were obtained for the percentage of plasmocytes and the number of antibody-producing cells. The investigator surmised that the lower maxima obtained for the group exposed for 12 weeks and immunized is an indication of adaptation to the RFR.

In another series, 12 rabbits were exposed to the same RFR at 5 mW/cm^2 for 6 months. Each month, the percentage of lymphoblast cells in peripheral blood was ascertained. The results showed an increase from about 3% initially to 9 and 10% respectively for months 1 and 2 of exposure, after which the percentage returned to baseline; these data also support the adaptation hypothesis. A smaller increase, to about 6%, was also seen for month 7 (1 month after cessation of exposure); values returned to baseline for months 8 and 9.

On day 6 of pregnancy, rats were exposed by Smialowicz et al. (1979a) to 2.45-GHz RFR for 4 hr/day, 7 days/week, at 5 mW/cm^2, until term. Following birth, pups were exposed until age 20 days, and half of these were exposed until 40 days of age. Equal numbers of pregnant rats and pups were sham-exposed for controls. The mean SARs for the pups diminished with age from 4.7 to 0.7 W/kg due to growth. Blood counts were made at ages 20 and 40 days, and the blastogenic responses of blood and lymph-node lymphocytes were determined by measuring the uptake of tritiated thymidine after stimulation of cell cultures with T- and B-cell mitogens. Two such experiments, each with a different exposure arrangement, were performed. The mean leukocyte counts at 20 days were found to be significantly lower for the RFR-exposed pups than for the controls in the first experiment but not in the second, and they were not significantly different at 40 days in either experiment. The results for the mitogen-stimulated cultures were widely scattered, and no consistent pattern was evident. Increases in thymidine uptake were seen in several cases, mostly for the cultures from the 40-day-old-rats; such results are difficult to interpret because the mean SAR had diminished by more than a factor of 2 since 20 days, and by a factor of 5 since the first few days of exposure.

Smialowicz et al. (1979b) also exposed mice to 425-MHz pulsed and CW RFR for 1 hr on each of 5 consecutive days. The power densities for the CW RFR were 39, 10, and 2.5 mW/cm^2; the corresponding SARs were 8.6, 2.2, and 0.55 W/kg, respectively. The average power densities for the pulsed RFR were 9, 2.5, and 0.63 mW/cm^2; the SARs were 2.0, 0.55, and 0.14 W/kg, respectively. No differences in the primary immune response to SRBC were found between mice exposed to the CW RFR and sham-exposed mice and between mice exposed to the CW and the pulsed RFR.

Pazderova-Vejlupkova (1979) used pulsed RFR at 2.74-GHz to expose rats for 4 hr/day, 5 days/week over 7 weeks. The average power density was 24.4 mW/cm^2, which caused a maximum rectal temperature rise of 0.5 deg C. Blood was taken before exposure, at weeks 1, 3, 5, and 7 during exposure, and at weeks 1, 2, 6, and 10 after exposure. There were significant decreases in leukocyte and absolute lymphocyte counts during the second half of the exposure period. In a similar experiment with 3.0-GHz pulsed RFR at 1 mW/cm^2 average power density, conducted in cooperation with the USSR Academy of Medical Sciences, the results were negative.

Hamrick et al. (1977) exposed fertile Japanese quail eggs for 24 hr/day to 2.45-GHz CW RFR at 5 mW/cm^2 (SAR about 4 W/kg), and reared the birds for 5 weeks after hatching. At this age, the levels of anti-SRBC antibodies were determined before, and 4 days after, challenge with SRBC. There were no significant differences between antibody levels for the quail from RFR-exposed eggs and control quail, either before or after challenge. In addition, no significant differences were found in the weights of the bursa of Fabricius (source of B lymphocytes in birds) or the spleen.

Shandala et al. (1979, 1977) exposed rats to 2.375-GHz RFR at power densities of 10, 50, or 500 microwatts/cm^2, 7 hr/day for 30 days and assayed blastic transformation of lymphocytes in mitogen-stimulated cultures on days 3, 7, 10, 14, 21, and 30. They reported a downward trend in the relative numbers of transformed T lymphocytes for 500 microwatts/cm^2. For the two lower power densities, they observed initial increases followed by decreases to less than control values. Autoallergenic activity was also reported for 500 microwatts/cm^2.

Guy et al. (1980) sham-exposed and exposed 4 rabbits each for 23 hr/day over 180 consecutive days to 2.45-GHz CW RFR. The power densities at the head and body were 10 and 7 mW/cm^2, respectively; the estimated peak SAR in the head was 17 W/kg, and the average SAR of the whole body was 1.5 W/kg. The eyes of the rabbits were examined periodically, and no differences were found between the two groups. Hematological (as well as other physiological) parameters were also evaluated, and the results for 0, 1.5, 3, 4.5, and 6 months of exposure were presented. No significant differences in values for RFR- and sham-exposed animals were found except for a decrease in the percentage of eosinophils (a type of leukocyte) at 6 months, but the investigators noted that this parameter varies widely among animals.

McRee et al. (1980) conducted hematologic and clinical-chemistry evaluations of the same animals (in another laboratory) shortly after completion of exposure and 1 month later, at which time the animals were euthanized and necropsies were performed. Again, the eosinophil percentage at completion of exposure was lower for the RFR- than sham-exposed rabbits; however, there was no depression 1 month after exposure. Regarding clinical-chemistry values, the only statistically significant results at completion of exposure were increases in albumin and calcium. However, 1 month after completion of exposure, the total globulin percentage had increased and the albumin percentage had decreased, both nonsignificantly, but the ratio of the latter to the former showed a statistically significant decrease. At necropsy, examinations of tissues showed no lesions attributable to RFR exposure. Samples of splenic tissue were cultured; stimulated with the mitogens PHA, Con-A, or pokeweed mitogen (PMW--a mitogen that stimulates both T and B lymphocytes), each in three different concentrations; and assessed for response by the uptake of tritiated thymidine. Lower responses for the RFR-exposed animals were obtained with all three mitogens, but the

differences were statistically significant only for PMW (at all three concentrations).

C.6.8.4 Health and Disease

Various other immunological responses of animals have been studied, including determinations of whether exposure to RFR alters the incidence or severity of disease. Such studies are difficult to conduct, and reliable, consistent results are hard to achieve.

Szmigielski et al. (1975) observed 5 rabbits that were experimentally infected with Staphylococcus aureus after exposure to 3-GHz RFR at 3 mW/cm^2 for 6 hr/day over 6 weeks, and 5 rabbits similarly treated but exposed for 12 weeks. They reported that the animals exhibited depression of peripheral-granulocyte counts and of granulocyte reserves mobilized by subsequent injection of Staphylococcus endotoxin, and increased lysozyme activity of serum. They stated that the animals appeared "sicker."

By contrast, Pautrizel et al. (1975) reported that exposure of mice to RFR (no frequency or intensity reported) conferred protection against an otherwise fatal challenge with Trypanosoma equiperdum. Also Prausnitz and Susskind (1962) observed that mice exposed to 9.3-GHz pulsed RFR at 100 mW/cm^2 for 4.5 min/day over 59 weeks appeared to have more resistance than controls to a pneumonia infection accidentally introduced into the colony; however, this was an incidental observation, not the results of a planned experiment.

On the other hand, Liddle et al. (1979) observed that mice immunized against Streptococcus pneumoniae had higher circulating antibody titers after exposure to 9-GHz RFR at 10 mW/cm^2, 2 hr/day for 5 days. However, this increase in antibodies failed to protect the mice against challenge with a virulent strain of S. pneumoniae.

Majde and Lin (1979) exposed mice to 148-MHz RFR at either 0.5 or 30 mW/cm^2 (SARs of 0.013 or 0.75 W/kg) for 1 hr/day for 3 successive days starting 24 hr after subcutaneous immunization with human type O red blood cells. Paw challenges for hypersensitivity were performed 14 days after immunization. The authors reported a mild but significant suppression of the anaphylactic response in the mice exposed at the higher but not at the lower power density. The degree of suppression of anaphylaxis was comparable to that seen in animals exposed to cold for 1 hr on the day following immunization, an interesting observation because exposure to cold is a known producer of stress.

Szmigielski et al. (1979) reported that exposure of mice to 2.45-GHz RFR at 20 mW/cm^2 for 2 hr/day, 6 days/week for up to 4 months and concurrent treatment with the carcinogens diethylnitrosamine or 3,4-benzopyrene led to earlier appearance of tumors than in unexposed

mice. However, exposure at 5 mW/cm^2 had no such "promoting" effect on carcinogenesis.

C.6.8.5 Summary and Conclusions on Immunological Effects

In summary, RFR does appear to have effects on the immune system of mammals. Some of the reported effects were obtained at low power-density levels, but most of the studies were performed at relatively high power densities, and in some cases effects obtained at high power densities were not found at lower power densities, suggesting the possibility that power-density thresholds exist. Some of the results indicate immunosuppressive effects, some indicate immunostimulative effects, and some indicate that the state of the immune system depends on the duration of exposure or when measurements were taken relative to the time of exposure or the time of day.

Effects on the immune system from chronic exposure to RFR at low power densities (tens to hundreds of microwatts/cm^2) are unlikely to be linked simply to temperature increases. The existing evidence indicates that some of the immune-system effects are probably mediated through the effect of RFR on the endocrine system, involving the general syndrome of adaptation to stress. The mechanisms and significance of such effects are not yet understood, nor have individual findings been independently verified. There is currently no evidence that reported RFR effects on the immune systems of animals at average power densities less than 1 mW/cm^2 would occur in humans exposed to the RFR from OTH-B outside the exclusion fence, or that such effects would be hazardous to human health. If chronic low-level RFR exposure did impair the ability to resist disease, then a relatively high rate of infectious diseases should occur among people occupationally exposed to RFR, and it is highly unlikely that this would not have been noticed. Possible low-level effects are taken seriously in the USSR because Soviet doctrine on environmental and occupational health (Zielhuis, 1974) holds that any measurable biological effect of any environmental agent is considered to be an unacceptable hazard to humans, regardless of its real medical significance.

C.6.9 Biochemical and Physiological Effects

The literature on biochemical and physiological effects associated with RFR is extensive. Many of the reported effects are associated with other events (e.g., changes in hormonal levels or stress adaptation), some are questionable for various reasons, and others do not have a clear medical significance.

C.6.9.1 In Vivo Studies

Among the relatively few investigations for possible effects of RFR on primates, and specifically with RFR in the HF range, was the study by Bollinger (1971). He exposed groups of 12 rhesus monkeys (Macaca

mulatta) each to 10.5- or 26.6-MHz pulsed RFR for 1 hr at average power densities of 200 or 105 mW/cm^2, respectively, or to 19.3-MHz RFR for 14 days, 4 hr/day, at 115 mW/cm^2. For each frequency, 12 monkeys were sham-exposed to serve as controls. Hematologic and blood-chemistry analyses were performed before and after exposure. The results indicated no statistically significant differences between exposed and control monkeys for most of the cellular components of the blood. Significant differences in mean monocyte and eosinophil counts were obtained, but were ascribed to conditions not related to RFR exposure. Similar conclusions were reached regarding the blood-chemistry parameters. Gross pathological and histopathological (microscopic) examinations of these animals showed no abnormalities ascribable to RFR exposure. In another part of this study, groups of 3 tranquilized monkeys each were exposed for successive time intervals at increasing power densities up to 600 mW/cm^2 for the two lower frequencies, and 300 mW/cm^2 for 26.6 MHz. Deep-body (esophagal) temperatures and electrocardiograms were taken during exposure. No obvious indications of thermal stress, increases of heart rate, or other influences on the electrical events of the heart cycle due to the RFR were found.

Frazer et al. (1976) exposed male rhesus monkeys to 26-MHz CW RFR at 500, 750, or 1,000 mW/cm^2 for 6 hr in a rectangular-coaxial-transmission-line chamber. Control monkeys were maintained just outside the exposure chamber. The ambient temperature was 22.2 deg C. Rectal and skin temperature were measured. At the highest power density, the skin and rectal temperatures increased during the first half hour by 2.5 and 1.3 deg C, respectively, and decreased during the next hour to 1.1 and 0.7 deg C above their respective initial values. The rectal temperature remained substantially constant during the remainder of the exposure period, but the skin temperature increased again slightly until the fifth hour and then declined during the last hour to 0.1 deg C above the initial value. Similar but smaller changes were obtained at the two lower power densities. These results indicate that even at the highest power density, the monkeys were in thermal equilibrium; i.e., they were able to dissipate the additional heat induced by the RFR, and their thermoregulatory mechanisms were quite efficient in doing so. Calculations by the investigators show that exposure of a 3.6-kg monkey to 26-MHz RFR at 1 W/cm^2 is approximately equivalent to exposing a human 1.8 m tall to this frequency at 400 mW/cm^2.

Krupp (1977b) performed similar experiments, exposing rhesus monkeys for 3 hr at frequencies of 15 and 20 MHz and power densities ranging from 0.76 to 1.27 W/cm^2. The results again indicated that the additional heat induced by the RFR is readily accommodated by the thermoregulatory mechanisms of the animals. Calculations show that exposure of a monkey to 20-MHz RFR at 1.27 W/cm^2 is equivalent to exposure of a human at 0.225 W/cm^2. The equivalence for 15 MHz at 1.025 W/cm^2 is 0.205 W/cm^2.

Krupp (1978) also did a follow-up study of 18 rhesus monkeys that had been exposed 1 to 2 years previously to 15-, 20-, or 26-MHz RFR for up to 6 hr on at least 2 occasions at power densities in the 0.5 to 1.27 W/cm^2 range. Hematological and biochemical blood parameters were measured, and physical (including ophthalmologic) examinations were performed. No variations from normal values or conditions that could be attributed to RFR exposure were found.

Adair and Adams (1980b) equilibrated squirrel monkeys for a minimum of 2 hr to constant environmental temperatures (22 to 26.5 deg C) cool enough to ensure that the cutaneous blood vessels in the tail and extremities were fully vasoconstricted (an effect produced by the thermoregulatory system to minimize heat loss). The monkeys then underwent 5-min exposures to 2.45-GHz RFR at successively higher power densities, starting at 2.5 to 4 mW/cm^2, until vasodilation in the tail occurred, as evidenced by an abrupt and rapid temperature increase for the tail skin. For example, a monkey equilibrated to 25 deg C exhibited tail vasodilation when exposed to RFR at 10 mW/cm^2 (whole-body SAR of 1.5 W/kg), whereas it did not when exposed to infrared radiation at the equivalent power density, an indication that the effect resulted from stimulation of thermosensitive elements of the thermoregulatory system by the RFR rather than heating of the tail skin. To cause tail vasodilation in monkeys equilibrated to lower environmental temperatures required RFR exposure at higher power densities. Specifically, an increase of 3 to 4 mW/cm^2 was found necessary for every 1 deg C reduction in environmental temperature.

The oxygen-consumption rate of an animal is a direct measure of its metabolic rate. Ho and Edwards (1977) used the oxygen-consumption rate as a biological indicator of stress. They exposed mice to 2.45-GHz RFR in a waveguide system that permitted continuous monitoring of the RFR absorption rate during exposure (Ho et al., 1973). The animals were exposed under the following controlled environmental conditions: 24 deg C temperature, 55% relative humidity, and 78 ml/min airflow rate. The forward power levels ranged from 0 to 3.3 W; the corresponding range of mean SARs was 0 to 44.3 W/kg. The exposures were for 30 min, during which the oxygen consumption and RFR absorption rates were determined at 5-min intervals; these values were converted into specific metabolic rates (SMRs) and SARs, respectively, expressed in the same units (W/kg). The oxygen-consumption rate was also measured at 5-min intervals for 30 min before and after exposure. Sham-exposed mice served as controls. At the highest forward power used, the SAR (averaged over 16 mice) decreased, during exposure, from 56 to 29 W/kg, and the SMR decreased from 17.5 to 14 W/kg, thereby decreasing the total thermal burden from about 74 to 44 W/kg. (The values for individual mice varied more widely.) Apparently the mice endeavored to decrease their thermal burdens by altering their body configurations during exposure to minimize their RFR absorption rates, and they reduced their oxygen consumption as well. Similar but smaller changes were obtained at forward powers of 1.7 and 0.6 W (mean SARs of 23.6 and 10.4 W/kg),

and insignificant changes were noted at 0.3 and 0.09 W (mean SARs of 5.5 and 1.6 W/kg). Thus, the onset of such RFR-induced thermal stresses corresponds approximately to the basal metabolic rate of the mouse (9 W/kg). Oxygen-consumption rates returned to normal after completion of RFR exposure.

Phillips et al. (1975) exposed rats to 2.45-GHz RFR for 30 min in a microwave cavity at SARs of 0, 4.5, 6.5, or 11.1 W/kg. Colonic temperatures were measured immediately after exposure, and measurements of colonic and skin temperatures, oxygen consumption, carbon dioxide production, respiratory quotient, and heart rate were recorded continuously for 5 hours, starting 10 min after exposure. Control rats were sham-exposed. The mean colonic temperature of the control rats immediately after sham exposure was 38.6 deg C, and it diminished gradually over the test period to a final value of about 38.0 deg C. For the rats exposed at 4.5 W/kg, there was an initial elevation of colonic temperature to 40.0 deg C, followed by a decrease to mean control values in 20 min and continuation at such levels for the remainder of the period. For those exposed at 6.5 W/kg, the initial colonic temperature increase was slightly larger (40.5 deg C) and was followed by a rapid decrease to levels significantly below control values that persisted for the remainder of the period. For the rats exposed at 11.1 W/kg, the initial colonic temperature value was much higher (42.4 deg C), but the temperature diminished more slowly, to values well below those for the 6.5-W/kg group by 3 hr after exposure, but it increased again to essentially control value by the end of the 5-hr period. Dose-rate-dependent elevations of skin temperature were observed shortly after exposure; temperatures diminished to normal values within 50 min. For the rats exposed at 4.5 and 6.5 W/kg, normal values persisted for the remainder of the period. However, for the group exposed at 11.1 W/kg, skin temperature continued to decrease during the next hour, finally leveling off at well below control values for the rest of the period. Oxygen consumption and carbon dioxide production by the 4.5-W/kg group were comparable to control values, but they were lower for the two groups exposed at the higher levels. Last, statistically insignificant bradycardia (lower heart rate) was observed in the 4.5-W/kg group; mild but statistically significant bradycardia developed within 20 min for the 6.5-W/kg group, which recovered within about 2 hr; pronounced bradycardia developed abruptly for the 11.1-W/kg group, after which heart rates increased to values well above those of controls (tachycardia), and persisted at these levels to the end of the test period. Irregular heart rhythms accompanied bradycardia, and incomplete heart block was evident for most of the rats exposed at the highest level, but the animals recovered within 60 min after cessation of exposure. Such heart block was surmised to be caused by the release of toxic materials, by elevated serum potassium, or by myocardial ischemia, all from excessive heat.

Han et al. (1978) exposed 3 rabbits dorsally and ventrally to 2.45-GHz RFR, CW or pulsed (1-microsecond pulses, 700 pps), at an

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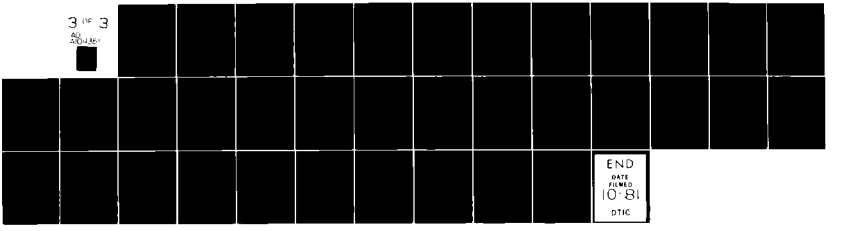

average power density of 5 mW/cm^2. For dorsal exposure at this power density, the maximum SARs were 0.86 W/kg in the brain and 0.09 W/kg in the heart; for ventral exposure, the corresponding values were 0.25 and 0.30 W/kg. The same animals were also exposed dorsally to 10-microsecond pulses, at a pulse power density of 13.7 W/cm^2, that were synchronized to the heart rate with 0-, 0.1-, or 0.2-s delay times relative to the R wave of the electrocardiogram. Before the experiments, the rabbits were acclimated for several weeks, and for at least 15 min immediately preceding exposure. Exposures were for 20 min, and heart rates were recorded every minute during exposure and for 15 min before and after exposure. No significant differences were observed between heart rates during the periods of exposure and nonexposure to RFR. The rabbits were later exposed at 80 mW/cm^2 CW RFR, which disturbed them sufficiently, because of the heat stress, to render heart-rate recording difficult. For this exposure, however, the heart rate increased, and returned to normal about 20 min after termination of exposure.

Moe et al. (1976) exposed 8 rats to 918-MHz CW RFR at 10 mW/cm^2 (mean SAR about 3.6 W/kg) for 10 hr/day over 3 weeks in cylindrical-waveguide chambers designed for chronic exposures under standard laboratory-maintenance conditions for rats. Eight sham-exposed rats served as controls. Physiological and behavioral comparisons between RFR- and sham-exposed rats showed no significant differences in fluid intake, body weight, rectal temperature, and corticosterone levels. However, food intake and blood glucose level were lower for the RFR-exposed animals, and their behavioral repertoires were altered, apparently to cope with the additional thermal burden imposed by the RFR.

Lovely et al. (1977) conducted a similar study but involving exposures to 918-MHz RFR at 2.5 mW/cm^2 for 10 hr/day over 13 weeks. No significant differences in behavioral repertoires, food intake, blood glucose, or most other blood-serum chemistry values were found. In another similar study by Lovely et al. (1979), performed with 2.45-GHz RFR at 5 mW/cm^2, the results were similar to those of Moe et al. (1976). The findings of these last three investigations are consonant with one another, given the SARs involved. They indicate the existence of an SAR threshold between 0.9 and 3.6 W/kg for such effects.

Deficis et al. (1979) exposed mice continuously for nearly 60 hr to 2.45-GHz RFR, either in unstirred multimodal cavities (12 mice simultaneously per cavity, with a total of 60 mice) at an estimated power density of 3.3 mW/cm^2, or in the nearfield of a slotted waveguide in an anechoic chamber (usually 12 simultaneously, with a total of 94 mice) at about 4 mW/cm^2. A total of 126 sham-exposed mice served as controls. Means of rectal temperature and body mass after treatment did not differ significantly for sham-, cavity-RFR-, and chamber-RFR-exposed groups. However, analyses of serum triglycerides (TRG) and beta-lipoproteins (LP) 2 hr after completion of exposure showed 30% higher mean TRG levels, than those for controls, for the mice

exposed in the cavities and 55% higher for the mice exposed in the chamber, an indication of RFR-induced stress. The mean LP levels were also 40% and 51% higher, respectively, than those for controls. Both sets of results show some dose-rate dependence. These findings may be related to those of Pazderova et al. (1974), discussed under "Epidemiology," who reported that workers in television and radio transmitting stations had decreased serum albumin levels and increased serum alpha- and beta-globulin levels. The report noted that although the changes were significant, the values were still within the normal human range and the workers appeared to be in good health.

Djordjevich et al. (1977) exposed rats for 90 days, 1 hr/day, to 2.4-GHz RFR at 5 mW/cm^2. Rectal temperatures were recorded before and after each exposure, and the animals were weighed daily. Blood samples were taken on day 10 before exposure; on days 30, 60, and 90 of the exposure period; and on day 30 after the period. These and samples from control rats were analyzed for total white cell count, erythrocytes, hematocrit, mean cell volume, and hemoglobin. Differential white cell counts for neutrophils, lymphocytes, monocytes, and eosinophils were also performed. Some rats were euthanized after the exposure period and their spleens, livers, hearts, brains, and testes were examined. No significant differences between RFR-exposed and control rats were seen for any of the analyses done or organs examined. Moderate leukocytosis was observed during the experimental period in both groups, with no significant differences between them, and the leukocyte levels returned to normal by day 30 after the exposure period. This effect was ascribed to seasonal responses by the rats.

Lin et al. (1979) sham-exposed or exposed mice to 148-MHz RFR at 0.5 mW/cm^2 (mean SAR of 0.013 W/kg) for 1 hr/day, 5 days/week for 10 weeks, starting on the fourth to seventh day after birth. Blood samples drawn at ages 28, 70, 100, 250, 300, 360, and 600 days (the first two immediately after exposure) were analyzed for hematocrit, hemoglobin, leukocyte count, erythrocyte count, and differential blood-cell counts. The results indicated that the formed elements in the blood were not affected by exposure to the RFR. The mice were also weighed daily during the 10-week treatment period and weekly thereafter until 600 days of age. Differences in weights between RFR- and sham-exposed mice at each age were not statistically significant.

C.6.9.2 Effects on Tissue In Vitro

Several published reports indicate that exposure of freshly excised tissues to RFR alters the functional activity of such tissues.

Lords et al. (1973) submerged isolated turtle hearts in Ringer's solution and exposed them to 96-MHz CW RFR, typically for 30 min, in a capacitor exposure system at applied powers in the range from 0 to 500 mW. Bradycardia was observed for the range from about 50 to 200 mW, and tachycardia at higher powers. They estimated that about 3.3% of the

applied power was absorbed by the heart, and that the temperature increase in the heart at 100 mW was about 0.2 deg C. These investigators also found that heating the solution (without RFR) produced tachycardia, so they hypothesized that the bradycardia observed in the lower power range resulted from RFR-induced neurotransmitter release by the remnants of the sympathetic and parasympathetic nervous systems in the heart preparation. In a later paper (Tinney et al., 1976), confirmation of this hypothesis was presented. The investigators showed that when propranolol hydrochloride (which blocks the sympathetic system) or atropine (which blocks the parasympathetic system) was added to the Ringer's solution, exposure to RFR did not produce bradycardia. Reed et al. (1977) produced bradycardia in isolated rat hearts exposed to the same frequency for 10 min in the SAR range from 1.5 to 2.5 W/kg; no bradycardia was observed when the same blocking agents were used.

McArthur et al. (1977) suspended post-pyloric segments of rat gut in Ringer's solution, permitted them to stabilize for 25 to 40 min at 28.5 deg C, then exposed them to 960-MHz RFR for 10 min at SARs of 1.5 to 5.5 W/kg. The waveform of peristaltic pressure of these smooth-muscle segments was monitored during stabilization and exposure, and for 30 to 45 min after exposure. Other similarly prepared segments were monitored while being maintained (without RFR exposure) in Ringer's solution with atropine as an additive, and still others were monitored during treatment with both atropine and RFR. Control segments were monitored for 90 to 120 min without either treatment. The results shown in Table 1 of the paper indicate that exposure to RFR alone significantly increased the rate of muscle contraction, whereas treatment with atropine alone caused a slight but nonsignificant decrease in peristaltic rate. Treatment with both atropine and RFR significantly decreased the peristaltic rate.

Whitcomb et al. (1979) suspended segments of rat gut in a modified Ringer's solution, permitted them to equilibrate for 1 hr at 7 deg C and for an additional hr at 36 deg C, then exposed them to 1-GHz RFR at SARs of 1.2, 2.3, or 6.9 W/kg. In contrast with the results of McArthur et al. (1977), exposure to RFR did not affect the rate of contraction. Differences in preparation and treatment methods may account for such contrary findings.

C.6.9.3 In Vitro Cellular Effects

Guy (1977) has described the development and characteristics of a transmission line cell-culture sample holder suitable for use in exposing a sample of cells in a culture medium for short periods to controlled broadband radiofrequency fields and controlled temperatures. Guy indicates that:

> In analyzing the data of many earlier experiments involving the effects of EM fields on cell cultures, blood samples and [illegible]

> concerning the exact magnitude of the fields and the temperatures of solutions during exposure. . . . Samples are often placed in fields of known strength and power density, but, due to the complex shape of vessels that hold the samples, the actual fields acting on the cells and the temperature in the sample are unknown. These unknowns make it difficult in many cases to determine whether observed effects are due specifically to the fields, or simply to a rise in temperature. (Guy, 1977)

These comments are relevant to evaluating the results and conclusions of the several papers reported in this section and other sections.

Michaelson (1970) made similar points with regard to evaluating studies on isolated cell systems, emphasizing that the interpretation of the biological results, e.g., cytogenetic effects, is difficult and does not necessarily lead to meaningful conclusions because of the many variables in tissue culture technique, e.g., influence of heat, viruses, and chemicals, that must be considered. In his most recent reviews, Michaelson (1980, 1978) has again emphasized the problems of interpretation of in vitro studies.

Riley et al. (1979) developed an experimental method for detecting "intrinsic" (as opposed to hyperthermic) effects of RFR on neoplastic (cancer) cells exposed in culture. They sham-exposed or exposed mouse lymphosarcoma cells to 30-MHz RFR at 100, 500, or 1,000 V/m (SARs of 31, 352, or 1,805 W/kg) in a Guy cell-culture system. Such cells were then implanted subcutaneously in mice of a specially selected immunocompetent strain. Tumors were produced with both RFR- and sham-exposed cells. However, a large percentage of the tumors derived from RFR-exposed cells subsequently regressed, resulting in host survival, whereas a large percentage of the tumors produced by the sham-exposed cells continued to grow, becoming lethal.

Baranski et al. (1974) reported increases in membrane permeability of rabbit erythrocytes and granulocytes during in vitro exposure for up to 3 hr to 1-GHz RFR at power densities of 1 to 10 mW/cm^2. Peterson et al. (1978) exposed rabbit and human erythrocyte cultures to RFR at 2.45 GHz or at swept frequencies of 12.5 to 13.0 GHz or 17.5 to 18.0 GHz. They found no significant differences in membrane permeability between cultures exposed at 10 mW/cm^2 and control cultures held at room temperature. However, comparable increases in permeability were obtained for cultures heated for 45 min with and without RFR to 37 deg C, indicating that such increases were thermally induced. Liu et al. (1979) obtained similar results. They exposed suspensions of rabbit, human, and dog erythrocytes for 3 hr to 2.45-, 3.0-, or 3.95-GHz RFR in a waveguide system at various SARs; the resulting temperatures ranged from 25 to 44 deg C. They also heated suspensions in a water bath to comparable temperatures. As a representative result, they found no significant differences in membrane permeability between suspensions

exposed to 3.0-GHz RFR at about 200 W/kg (which corresponds to an equivalent plane-wave power density of about 42 mW/cm^2 at the center of the waveguide) and suspensions heated by water bath to the same temperature (44 deg C). Likewise, Janiak and Szmigielski (1977) reported no significant differences in the sequence and time course of mouse fibroblast cells heated to 43 deg C in a water bath or by exposure to 2.45-GHz RFR.

Corelli et al. (1977) investigated the effects of 2.6 to 4.0 GHz RFR on colony-forming ability (CFA) and molecular structure (determined by infrared spectroscopy) of Escherichia coli B bacterial cells in aqueous suspension. Cells were exposed for 10 hours at an SAR of 20 W/kg (estimated to be approximately equivalent to 50 mW/cm^2 plane-wave exposure). No RFR-induced effects on either CFA or molecular structure were observed.

Other reported effects of in vitro exposure of cells to RFR are the calcium-efflux phenomenon, alterations of the blood-brain barrier, and changes in leukocyte proliferation, differentiation, and functional capacity These topics are discussed in Sections C.6.5.2, C.6.5.3, and C.6.8, respectively.

C.6.9.4 Conclusions Regarding Biochemical and Physiological Effects

The thermal basis for the reported in vivo biochemical and physiological effects of RFR is evident. Most significant are the investigations with nonhuman primates because of their close anatomical and physiological similarities to humans, and especially relevant to OTH-B are the results of exposures of rhesus monkeys at frequencies in the HF range (Bollinger, 1971; Frazer et al., 1976; Krupp, 1977b). These results showed that exposures to average power densities exceeding 100 mW/cm^2 were within the thermoregulatory capabilities of these animals. Also noteworthy were the negative results of the blood-chemistry assays performed on rhesus monkeys 1 to 2 years after such high-power-density exposures (Krupp, 1978). The investigations with other species exposed at higher frequencies (148, 918, and 2450 MHz) and lower power densities yielded a variety of positive and negative results. However, none of the results indicates that exposure of humans to the levels of RFR from OTH-B outside the exclusion fence will induce any detectable biochemical or physiological effects.

For exposures of isolated tissue preparations in vitro to RFR, it is more meaningful to cite SAR values (if measured or estimated) than incident power densities. The lowest SAR at which bradycardia was observed in the isolated turtle heart was 1.5 W/kg (Reed et al., 1977). Coincidentally, this was also the lowest value at which McArthur et al. (1977) induced increases in the contraction rate of isolated muscle segments (a finding that was not confirmed by Whitcomb et al., 1979, for SARs up to 6.9 W/kg). Even if these two positive findings are accepted at face value, they do not constitute evidence that such effects would

occur in humans from exposure to the RFR from OTH-B outside the exclusion fence because the SARs there are considerably less than 1.5 W/kg. The same conclusion is applicable to the in vitro cellular effects discussed above, which were obtained at much higher SARs than [illegible] in the tissue preparations.

C.7 Misconceptions

Several misconceptions regarding the bioeffects of RFR continue to be expressed in popular accounts outside peer-reviewed scientific publications on the subject. Those accounts tend to be sources of some confusion for the nonspecialist. The following are representative examples.

The distinction between RFR and ionizing radiation is often not made; consequently, the known hazards of the latter are linked--by implication--with exposure to RFR. In essence, ionizing radiation (which includes ultraviolet light, X-rays, and the emissions from radioactive materials) has sufficient quantum energy (see Section C.5.1) to expel an electron from a molecule, leaving the latter positively charged and thereby strongly affecting its interactions with neighboring molecules. Ionization can alter the functions of biological molecules fundamentally and often irreversibly.

By contrast, the quantum energies of RFR are so much smaller that their primary effect is to agitate molecules rather than to ionize them. (The possibility of long-range quantum interactions, discussed in Section C.5.1.3, is not excluded; however, evidence of their occurrence in live animals is sparse as yet, and there is no evidence that such effects would be harmful if they do occur.) Also, RFR-induced agitation ceases as soon as exposure to RFR is halted. At low RFR intensities, the heat that such agitation represents is well accommodated by the normal thermoregulatory capabilities of the biological entity exposed, and therefore such effects are generally reversible. At high RFR intensities, the thermoregulatory capabilities may be inadequate to compensate for such effects, and exposure at such intensities may lead to thermal distress or even irreversible thermal damage. In short, a single quantum of ionizing radiation that is absorbed by a molecule alters the properties of that molecule, and exposure to such radiation may thereby profoundly affect the function of the biological constituent involved, whereas the concurrent absorption of many quanta of RFR is necessary to cause biologically significant effects.

Even if an effect is produced by RFR, that effect may not necessarily be deleterious to the entity involved. As an example of a nonhazardous biological effect, the absorption of visible light (a form of electromagnetic radiation having quantum energies above those of RFR but below those of the ionizing radiations mentioned previously) in the eyes is necessary for vision. Light is also absorbed by the skin and at normal levels is converted into harmless heat. One of the reasons that

the levels of allowable exposure of humans to RFR are generally lower in Eastern European countries than they are in the West is the philosophically based assumption that every physiologically detectable effect of RFR is potentially harmful--a view not generally shared in Western countries.

Concerned people often ask whether guarantees can be offered that chronic exposure to low levels of an agent such as RFR will have no deleterious effects many years in the future. It is scientifically impossible to obtain data on which a guarantee of absolute safety can be based. However, the large body of experimental data on the bioeffects of RFR indicates that, unlike the ingestion of certain substances in small quantities that can accumulate into a potentially harmful dose, RFR energy continually absorbed at low incident power densities (dose rates) is readily dissipated and does not accumulate in the body toward the equivalent of RFR energy absorbed at high incident power densities. This is one of the basic reasons for the existence of threshold power densities for the various RFR bioeffects.

C.8 Unresolved Issues

The potential biological effects of RFR from OTH-B have been assessed from existing studies at frequencies up to 18 GHz. Based on the studies evaluated, with recognition that the negative findings reported in some studies may have been obtained because the experiments had been poorly conducted, there is no evidence that exposure to the RFR from the OTH-B transmitter outside the exclusion fence will be hazardous to human health. However, certain gaps remain in our knowledge of the biological effects of RFR. These gaps may be identified as follows:

(1) Available results from animal studies and mathematical models are insufficient to permit adequate predictions regarding similar biological effects in humans. Moreover, most animal research has not involved continuous exposure over periods approaching an animal's lifetime. These deficiencies affect the interpretation of experiments done at different RFR frequencies and the interpretation of differences in biological response among various species.

(2) Prospective epidemiological studies of effects of exposure of humans to RFR are lacking. Existing epidemiological studies, while extensive and reasonably well-done, are all retrospective in nature, and are therefore subject to inherent defects of method.

The probability that new information would reveal a significant hazard cannot be dismissed, but is judged to be relatively low.

C.9 OTH-B and Safety to Human Populations

In the previous sections, the current state of knowledge regarding the biological effects of RFR was examined on a topic-by-topic basis. Representative articles relevant to OTH-B were selected from the large body of scientific literature for review and analysis. The discussions also covered related topics, such as background information on other RFR-emitting devices and equipment in the United States; safety standards in the United States and other countries; problems of risk assessment, related to scientific issues, philosophical positions, and range of legal applicability of safety standards; mechanisms of interaction of RFR with biological entities, involving definitions of "thermal" and "nonthermal" and distinctions between interactions of CW and pulsed RFR; uncertainties in retrospective epidemiological studies; and the basic problems of assessing possible hazards to humans of any environmental agent by extrapolating results of experimental research performed on animals. In this subsection, the evidence and conclusions regarding whether operation of OTH-B would constitute a possible hazard to humans are summarized.

Most U.S. experiments with animals that yielded recognizable and repeatable effects of exposure to RFR were performed at incident average power densities of more than about 2 mW/cm^2. Such effects are thermal, in the sense that the RFR energy is absorbed by the organism as widely distributed heat that increases the whole-body temperature, or as internally localized heat that is biologically significant even with functioning natural heat-exchange and thermoregulatory mechanisms operating. The existence of threshold average power densities has been experimentally demonstrated for some effects and postulated for the others. Exposure to RFR at average power densities exceeding the threshold for a specific effect for durations of a few minutes to a few hours (depending on the value) can cause irreversible tissue alterations. The heat produced by indefinitely long or chronic exposures at power densities well below the threshold is not accumulated because its rate of production is readily compensated for by heat-exchange processes or thermoregulation. Most investigations involving chronic exposures of mammals yielded either no effects or reversible, noncumulative behavioral or physiological effects for average power densities exceeding 2 mW/cm^2. In the few cases in which irreversible adverse effects of exposure were found, such effects were absent for average power densities below 2 mW/cm^2.

Few experiments show any biological effects of RFR at incident average power densities less than about 2 mW/cm^2. Such effects are often called "nonthermal," to distinguish them from those considered above. However, this usage of "nonthermal" is confusing and imprecise because the interaction mechanisms involved in each such effect differ considerably from those for the other effects, and clear distinctions between "thermal" and "nonthermal" based on precise scientific definitions of these terms are difficult to discern in the

interactions. Among the so-called nonthermal effects of RFR that have been documented to date are the RFR auditory phenomenon and the calcium-efflux effect. However, because OTH-B will emit FMCW RFR rather than pulsed- or amplitude-modulated RFR, neither phenomenon is relevant to OTH-B operation. Moreover, no known effects have been attributed to the frequency modulation per se of FMCW RFR.

Collectively, the results of the relatively few retrospective epidemiological studies performed in the United States, the USSR, and other Eastern European countries are not regarded as evidence that the RFR from OTH-B is likely to constitute a hazard to the general population.

In short, no reliable evidence has been found to support the conclusion that any hazard will result from either short-term or long-term exposure of people outside the exclusion fence to the RFR from OTH-B.

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